"I would have everie man write what he knowes and no more."—MONTAIGNE

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EDITORIAL

THE blessing of anaesthetics to suffering humanity can hardly be exaggerated. The agony that many endured for want of them is too horrible to contemplate. The development of modern surgery would have been impossible without them. Nevertheless, though there can be no belittling of the benefit of anaesthesia, no single anaesthetic or combination of anaesthetics but leaves something to be desired. Years ago many surgeons insisted on chloroform for their abdominal sections; it had no rival for the pains of childbirth. It held the field for a longer time than any other anaesthetic has ever done, but it is rarely used now. Ether took its place in large measure but has seen its field of usefulness much curtailed especially of late years. Nitrous oxide, often proclaimed as the safest of all anaesthetics, is often inadequate and when not mixed with sufficient oxygen has caused not only death but irreparable damage to the brain. Spinal anaesthesia, which can show results as good as any and better than most, waxes and wanes, is unsuitable for operations on the upper half of the body, has been followed by dramatic tragedies and in consequence is out of favour at the present time. Curare and other synthetic compounds have largely taken its place because they alone can rival the muscular relaxation produced by a spinal anaesthetic. But as these drugs are merely relaxants and not anaesthetics, additional methods have to be used to render the patient oblivious to pain. This combination of relaxant and anaesthetic has many advantages and at the present moment is the most popular way of rendering a patient ready for an operation. Already, however, reports have come from America that there are six times as many deaths following the use of curare as when one of the older techniques is used. British anaesthetists do not for the most part subscribe to this opinion, but the number of people who die unexpectedly after curare has been given seems to be on the increase. Should further experience establish this as fact it will be necessary to find the reason, whether it lie in some property inherent in the drug itself or in its misuse owing to our ignorance.

IN this number of the Journal there are two reports on the new fluorinated compounds used for anaesthesia: "Fluoromar," a fluorinated ether, and "Fluothane," a halogenated hydrocarbon. "Fluoromar" seems remarkable among anaesthetics in that adrenalin can be injected in animals under its influence without disturbance of the cardiac rhythm. For its other qualities, as ascertained by the authors of the paper, we refer our readers to the summary. "Fluothane," of which Dr. Michael Johnstone gives his experience in some 500 cases, seems more promising. In the capable hands of Dr. Johnstone it has made an excellent start. Details of his method will be found in his article. The outstanding features of his report on the action of "Fluothane" are as follows:

(1) The great variety of serious cases successfully anaesthetized.

(2) The complete absence of salivary, mucous and bronchial secretion.

(3) The absence of nausea and vomiting in over 90 per cent of the cases.

(4) Smooth and rapidly reversible anaesthesia.

(5) Last and most remarkable of all, the complete absence of shock. The rapid pulse, the cyanotic pallor, the cold clammy skin so characteristic of that condition were never seen, being replaced by a skin "Warm, dry and pink." See case 3, for example.

What place either of these compounds will come to occupy in the hierarchy of anaesthetics future experience will no doubt decide.

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THE HUMAN CARDIOVASCULAR RESPONSE TO FLUOTHANE* ANAESTHESIA

BY

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TRIFLUOROETHYL vinyl ether (Fluoromar) was the first fluorinated volatile compound to be used in clinical anaesthesia (Krantz, et al., 1953; Orth and Dornette, 1955). Its lower limit of inflammability was 3 per cent of the vapour. Prior to this a series of fluorinated compounds was investigated by Robbins (1946) who observed that the anaesthetic potency of a fluorinated compound was greatly increased by the insertion of a second halogen atom. This work has been extended by Raventós and Suckling (1956) and the compound CF_3-CHClBr (2 : bromo 2 chloro 1 : 1 : 1 : trifluoro ethane) was found to have excellent anaesthetic properties in animals as well as being nonexplosive and noninflammable when mixed in high concentrations with oxygen. This compound will be referred to as Fluothane.

Fluothane is a colourless liquid with a specific gravity of 1.86 at 20° C. and its boiling point is 50.2° C. at 760 mm Hg pressure. Its vapour pressure is 439 mm Hg at 20° C. Vapour concentrations within the anaesthetic range have a pleasant and nonirritant odour. The compound is decomposed by light but is stable if stored in amber-coloured bottles ; the action of light is prevented by thymol 0.01 per cent w/w. Decomposition does not take place in the presence of warm soda lime.

The pharmacology of Fluothane has been investigated in detail by Raventós (1956). It is about four times as potent as di-ethyl ether with a therapeutic ratio about twice that of ether. Surgical anaesthesia can be rapidly induced in animals with from 2 per cent to 4 per cent of the vapour in oxygen and maintained for several hours on 1 per cent with excellent relaxation and spontaneous respiration. Recovery of consciousness is rapid—two to ten minutes—even after prolonged anaesthesia. Salivary and mucous secretions were abolished in nonatropinized animals and neither retching nor vomiting occurred during induction, maintenance, or recovery from anaesthesia. Cardiac irregularities did not appear during any phase of anaesthesia though ventricular tachycardia and ventricular fibrillation were precipitated by the intravenous injection of adrenaline and noradrenaline to dogs anaesthetized with the drug.

A consistent finding in most animals was a fall in blood pressure which was directly proportional to the concentration of Fluothane vapour inhaled. This effect has been related to a block of the sympathetic ganglia with splanchnic vasodilation. The hypotensive effect was minimal

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with the 1 per cent vapour concentration used for maintenance.

Histopathological studies revealed minor cytological changes in the livers and kidneys of experimental animals after prolonged Fluothane anaesthesia. These changes did not appear to be associated with significant functional or biochemical alterations.

In view of the rather attractive pharmacological properties of this drug a clinical trial was begun. This paper is a report on the effect of Fluothane on some 500 patients. The list of operations performed on them is given later. Some of the patients would come under the heading of "good risks", whilst others could be described as "grave risks" and a still greater number would fall into intermediate categories. This initial report will be concerned mainly with the cardiovascular effects of Fluothane when administered from a Boyle's apparatus.

METHOD

The smaller (Trilene) bottle of a Boyle's apparatus was found to provide a vapour concentration of from 0.2 to 4.2 per cent at 25° C. with a gas flow of 10 litres a minute, Fluothane filled to the 100ml mark, and the plunger fully withdrawn. Under similar circumstances with 50 ml of Fluothane in the bottle the vapour range was 0.1 to 2.8 per cent. With the plunger fixed at 3 mm above the surface of the liquid the vapour range was 0.6 to 4.8 per cent.

It is obvious that only approximate values of vapour concentrations can be obtained when using this equipment for prolonged anaesthesia. In recording the strength of Fluothane vapour used in the clinical trial I will refer to a mark on the extended scale (fig. 1B) which was fixed alongside the control lever of the bottle and gives the approximate vapour concentration as calculated from a chart provided for this particular vaporizer. In all cases the 10-litre gas flow remained constant and consisted of 5 litres of oxygen and 5 litres of nitrous oxide; the plunger was always fully withdrawn; and the liquid maintained approximately at the 50 ml mark.

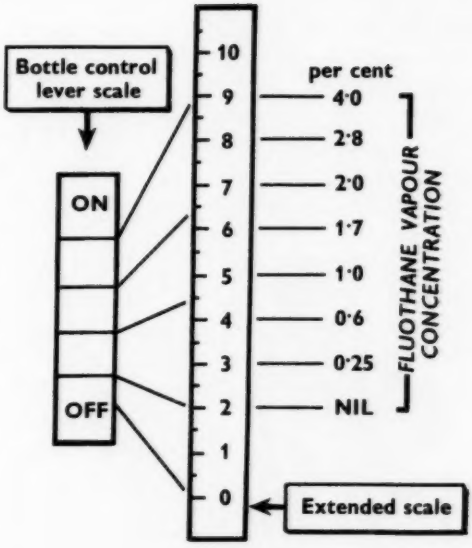

FIG. 1A

Diagram of the extended scale showing the vapour concentrations available at 25°C. with 100 ml of Fluothane in the bottle, the plunger fully withdrawn, and a gas flow of 10 litres a minute.

Blood pressures were estimated by sphygmomanometry and radial pulse palpation in all cases before induction and at frequent intervals thereafter. Respiratory rates were recorded at frequent intervals. Continuous electrocardiography was used

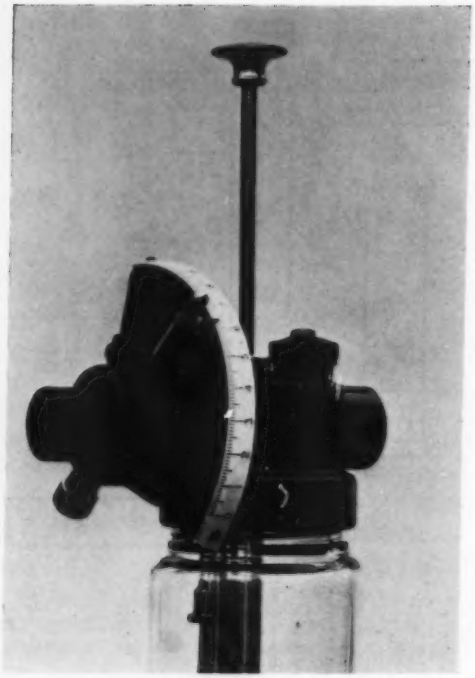

FIG. 1B
Photograph of vaporizer showing extended scale fixed alongside the control lever.

during most major operations; cardiograms were obtained of disturbances of rhythm which occurred before or during the administration of the anaesthetic.

Premedication.

Atropine only, 0.6 mg thirty minutes before induction ..... 108 cases
Atropine 0.6 mg with pethidine 50 mg before induction ..... 392 cases

Induction of Anaesthesia.

In all cases, except where otherwise stated, anaesthesia was induced with a sleep dose of thiopentone (250 mg) injected intravenously followed by 50 mg of suxamethonium and oropharyngeal or orotracheal intubation. The induction of anaesthesia with Fluothane will be made the subject of a separate study and report.

Series One. Vapour concentration 2.2 to 3.2 per cent approximately; lever mark 7 to 8; plunger fully withdrawn; 50 ml of Fluothane in the bottle.

This initial series consisted of fifty adults with normal cardiovascular and respiratory systems undergoing operations such as herniorrhaphy, mastectomy, thyroidectomy (nontoxic), varicose vein, and various orthopaedic procedures. Each operation lasted a minimum of twenty-five minutes and took place with the patient in the supine position. Each patient was premedicated with atropine 0.6 mg and pethidine 50 mg and an airway inserted under the thiopentone-suxamethonium sequence. Respiration was then controlled with oxygen, nitrous oxide and Fluothane (mark 7 or 8) until the return of spontaneous respiration a few minutes later. Anaesthesia was then maintained with a similar mixture till the end of the operation.

Smooth anaesthesia persisted throughout the period of operation in all cases, without narcotic or relaxant supplementation. Adequate relaxation was always present. The respiratory rate tended to increase during Fluothane anaesthesia and particularly with surgical stimulation, the average rate during the operations being 28 respirations a minute. The transition from suxamethonium apnoea to spontaneous respiration under Fluothane anaesthesia was made without coughing or straining. In each case reflex activity returned within two or three minutes of

stopping the anaesthetic and was followed by a five- to ten-minute period of apparently natural sleep from which the patient resented being roused. Full consciousness then returned and was not associated with any restlessness, retching, or vomiting; none of the patients complained of nausea.

The administration of Fluothane vapour in this concentration range caused a rapid fall in blood pressure within two or three minutes of starting. In 35 cases the systolic pressure dropped to between 60 and 80 mm Hg and the hypotension was associated with brisk vasodilatation with an increase in the volume of the peripheral pulse; the skin became warm, dry, and pink, and remained in this state throughout the period of anaesthesia. There was no capillary oozing from the cut tissues though the larger vessels bled normally.

As the blood pressure fell during Fluothane anaesthesia the pulse rate invariably became slower and rates as low as 45 beats a minute were observed. Atrioventricular nodal rhythms were seen in five patients, atrioventricular dissociation with interference in two, and an unusual form of sinus arrhythmia with coupling of sinus beats was seen in two patients (fig. 2B). In all these cases sinus rhythm was restored by the intravenous administration of atropine 0.5 mg. This dose of atropine also raised the systolic pressure from 60 to 80 mm Hg in three patients.

In most cases the hypotensive effect persisted throughout the period of anaesthesia. In eight cases the systolic pressures, after initial falls to 80 mm Hg, rose gradually to 100 mm Hg and persisted at this level for the remainder of the operations.

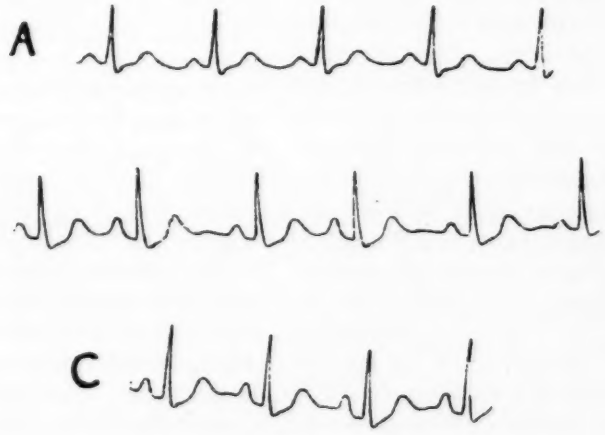

FIG. 2

Male, 48 years. Fit and muscular. Nephrectomy for stone. Premedicated with atropine 0.6 mg with pethidine 50 mg. All Lead 2.

A. Before induction.

B. After thirty minutes Fluothane (scale mark 8) anaesthesia; note the unusual coupling of the sinus beats.

C. Thirty seconds later, after atropine 0.5 mg intravenously.

In nine patients the hypotension outlived the narcosis by twenty minutes. There were no increases in pulse rates during the recovery periods, and in several patients the rates tended to decrease as the blood pressures returned to normal.

Series Two. Vapour concentration 0.75 to 1.25 per cent approximately; lever mark 6 to 7; plunger fully withdrawn; 50 ml of Fluothane in the bottle.

Fifty patients, with normal cardiovascular and respiratory systems, undergoing operations similar to those in the previous series. Each patient was premedicated with atropine 0.6 mg and pethidine 50 mg administered intramuscularly thirty minutes before induction. An additional dose of 0.5 mg of atropine was mixed with the sleep dose of thiopentone, suxamethonium 50 mg injected and an airway inserted. Anaesthesia was then maintained with oxygen, nitrous oxide 50 per cent, and Fluothane (1 per cent approximately). This concentration provided smooth and even anaesthesia with adequate spontaneous respiration and sufficient relaxation for all the operations. The respiratory rates increased to an average of 30 a minute; this rate could be reduced to any required level by the intravenous administration of pethidine in doses up to 40 mg. The recovery phases were similar in all respects to those in the previous series, except that one patient vomited a few times and two complained of nausea. The patient who vomited stated that she became nauseated shortly after the pethidine was administered pre-operatively and she vomited twice before the induction of anaesthesia. During cold weather it was noted that several patients shivered for a few minutes after recovering consciousness; the shivering required no specific treatment.

The incidence of hypotension was considerably less in this series. The systolic pressures fell to 70 mm Hg in six patients and in the remainder the average systolic pressure during the maintenance of anaesthesia was 100 mm Hg. The mild hypotension associated with Fluothane (1 per cent approx.) anaesthesia persisted throughout the period of anaesthesia in most cases, but in sixteen patients the systolic pressures gradually returned to within normal limits after thirty minutes anaesthesia. A perfectly regular sinus rhythm persisted throughout anaesthesia in all cases, the average rate being 84 beats a minute. Heart rates in general tended to decrease slightly as the operation progressed, and there were no reactionary tachycardias in any patients during recovery from anaesthesia. Throughout the period of anaesthesia in each case the skin remained warm, dry, and pink, with prominent superficial veins. The cardiograms showed no significant changes in the ST segments or T waves.

PRELIMINARY CONCLUSIONS

The results obtained in this series indicated that Fluothane provided very smooth and easily reversible anaesthesia with adequate relaxation for the operations selected. Although relaxation was of a high degree there was no obvious evidence of respiratory depression with the vapour concentrations used. The effects on the cardiovascular system—hypotension and bradycardia—were consistent with a depression of sympathetic activity with sparing of the cardiac parasympa-

thetic mechanism—a syndrome very similar to that induced by high spinal analgesia. Other signs suggestive of depression of autonomic activity were the persistent vasodilatation, the complete absence of sweating, the complete suppression of salivary, bronchial, and gastric secretions, and the absence of vomiting and retching in the recovery period.

It is obvious that the intensity of the cardiovascular reaction to Fluothane anaesthesia is directly related to the vapour concentration used, being minimal with the 1 per cent which appears to be the concentration needed for maintenance in most cases. Many of the patients felt quite hungry and were able to enjoy substantial meals within two or three hours of the operations. There were no obvious signs of postoperative damage to kidneys, livers, or lungs, though the effects of Fluothane on the functions of these organs will be the subject of more detailed investigations.

In view of the encouraging results obtained in the preliminary studies, the use of Fluothane was immediately extended to cover most types of surgical operations on patients with either normal or abnormal cardiovascular and respiratory systems. The only patients excluded were those with obviously diminished blood volumes pre-operatively. An additional 400 cases have now been completed, their ages ranging from 7 months to 86 years. Premedication was either atropine 0.6 mg alone or combined with pethidine 50 mg; anaesthesia was induced with a sleep dose of thiopentone (unless otherwise stated) and an airway inserted under suxamethonium paralysis. The additional 0.5 mg of atropine was usually injected with the thiopentone and omitted only when tachycardia was present or when hypotension was considered to be necessary to limit bleeding, e.g. neurosurgery, mastectomy, etc. Fluothane was used in the vapour concentration range of 0.75 to 1.25 per cent (scale marks 6 to 7) with nitrous oxide and oxygen 50 per cent. Details of the operations for which Fluothane was the main anaesthetic used are as follows:

Abdominal Surgery	139	cases
Cholecystectomy	28	
Cholecyst-duodenostomy	7	
Gastrectomy, partial	23	
Gastro-enterostomy, with vagotomy	15	
Hemicolectomy	12	
Splenectomy	6	
Adrenalectomy, bilateral	4	
Colostomy	12	
Appendicectomy	27	
Resection of rectum, abdoperineal	5	
Genito-urinary Surgery	129	cases
Prostatectomy, suprapubic	46	
Prostatectomy, per urethral	24	
Cystoscopy, with diathermy, etc.	37	
Nephrectomy	11	
Nephrolithotomy	8	
Nephrostomy	1	
Diverticulum of bladder	1	
Cystectomy, total	1	
Orthopaedic Surgery	31	cases
Compression screws, neck of femur	2	
Trauma	3	
Fractures, reduction and fixation	10	
Amputations	16	
Herniorrhaphy	23	cases
Neurosurgery	29	cases
Craniotomy	15	
Laminectomy	6	
Radiography	5	
Carotid ligation	3	
Mastectomy (radical)	17	cases
Thyroidectomy	23	cases
Toxic	16	
Nontoxic	7	
Gynaecology	4	cases
Caesarean section	1	
Colporrhaphy	3	
Chest Surgery	5	cases
Mitral valvotomy	3	
Cardiac neurectomy	2	

Fluothane provided excellent relaxation for all these cases with the exception of the laparotomies, some of which required supplementation with relaxants. Adequate spontaneous respiration was present throughout the operations and seldom required any assistance. In the majority of patients the administration of Fluothane caused an increase in the respiratory rate, the depth remaining either unaltered or slightly increased. Surgical stimulation, particularly in the region of the anus, often caused an increase in the respiratory rate, and rates up to 60 a minute have been encountered during haemorrhoidectomy. These rapid rates were not associated with any increase in the resistance of the lungs to inflation nor was there any increase in the tone of the abdominal musculature such as occurs during trichlorethylene tachypnoea. Tachypnoea not associated with anal stimulation can be controlled with pethidine in doses up to 40 mg; that associated with anal stimulation is effectively abolished by suxamethonium.

Brisk reflex activity returned in two or three minutes after stopping the Fluothane in all cases and vomiting during recovery was present in about 4 per cent of patients, being most evident after cholecystectomy. Transient postoperative nausea was present in 8 per cent of the patients.

Fluothane in the concentration range of 0.75 to 1.25 per cent caused a moderate drop in blood pressure in most cases and a drop to 90 mm Hg in 10 per cent of cases. After twenty or thirty minutes anaesthesia the systolic pressure gradually returned to within normal limits in about half of these patients. Anaesthetized patients were often sensitive to changes in posture, and the reversed Trendelenburg position sometimes precipitated a profound hypotension. Sudden loss of blood during anaesthesia was associated with a sudden fall in the systolic pressure without much change in the pulse rate; the reaction to the transfusion of blood or saline was rapid. Traction on abdominal viscera or retraction of brain tissue caused sudden drops of blood pressure with slowing of the pulse rate in about 20 per cent of the patients subjected to these manœuvres.

Essential hypertension, which was present in eighteen patients, caused no significant alterations in the cardiovascular reactions to Fluothane.

The hypotension associated with Fluothane anaesthesia can be prevented or immediately abolished by the intravenous injection of methoxamine hydrochloride (Cummings and Hays, 1956) in 5-mg doses. This drug has invariably produced the desired effect and is much more consistent in its vasopressor action than other drugs of this type. It has not caused any cardiac arrhythmias or tachycardias when administered to patients anaesthetized with Fluothane. The dose should be carefully restricted, as excessive amounts—10 mg and over—may cause severe hypertension. Methoxamine completely prevents the hypotension associated with the head-up position.

Cardiac Rhythm during Fluothane Anaesthesia.

Sinus rhythm has persisted at a steady rate throughout anaesthesia in most cases. Atrioventricular nodal rhythm was extremely rare, probably as the result of the

additional atropine administered with the thiopentone during the induction of anaesthesia. The sinus rate has seldom shown any tendency to increase even after periods of anaesthesia up to 270 minutes nor has there been any increase during and after the recovery of consciousness. Ventricular extrasystoles were observed in twelve cases and were either of the isolated variety or coupled with sinus beats (bigeminy); in each case the arrhythmia was associated with impairment of ventilation either as the result of positioning—steep Trendelenburg or lateral positions—or by respiratory depression from relaxants, and in each case was quickly abolished by assisting the respiration.

The operations of thyroidectomy for thyrotoxicosis and bilateral adrenalectomy for carcinomatosis have not been associated with any disturbance of cardiac rhythm during Fluothane anaesthesia. The application of the electric cautery to haemorrhoids and to bleeding points in the prostatic bed has precipitated transient sinus tachycardia with isolated ventricular extrasystoles in four patients, the cardiovascular disturbance being associated with brisk tachypnoea. The reactions have not been observed during Fluothane anaesthesia when the nerve supply to the region has been blocked by a "saddle-block" spinal analgesic.

The shock syndrome—sinus tachycardia, hypotension, vasoconstriction, sweating, and delayed recovery of consciousness—has not been seen in any patient either during anaesthesia with Fluothane or after the recovery of consciousness. Delayed circulatory collapse —24 to 48 hours postoperatively—occurred in three patients after major surgery and is suspected to be related to hypokalaemia.

Relaxants and Fluothane.

The use of relaxants to facilitate the exploration of the peritoneal cavity is probably advisable when using Fluothane anaesthesia. Attempts to produce with Fluothane in all cases the complete paralysis of the abdominal musculature nowadays expected by the surgeons may result in unnecessarily severe degrees of hypotension requiring postural adjustments.

In the initial stage of this part of the investigation 15 mg of d-tubocurarine were injected intravenously into each of ten patients anaesthetized with Fluothane (scale mark 7); laparotomy for appendicitis was being performed on each and the d-tubocurarine was injected as the peritoneum was being incised. Following the injection of d-tubocurarine in each case there was an immediate drop in blood pressure to between 60 and 70 mm Hg, without significant change in the pulse rate.

Controlled respiration was then necessary in three patients, and in one of these the peripheral pulse disappeared completely during positive pressure inflation of the lungs, and a cyanotic pallor developed; the slightest pressure on the rebreathing bag obliterated the carotid pulse which returned slowly and sluggishly when the pressure was withdrawn. The appendicectomy was hurriedly completed and neostigmine 2.5 mg with atropine 0.5 mg were administered intravenously. As soon as spontaneous respiration commenced there was a dramatic im-

provement in the patient's circulation: the systolic pressure rose to 70 mm Hg and the pulse was easily palpable at the wrist. Three minutes later a severe bradycardia with hypotension recurred—neostigmine effect—and was modified by a further 0.5 mg of atropine; consciousness returned in fifteen minutes and the systolic pressure became normal two hours later; subsequent recovery was uneventful. Similar syndromes of somewhat less alarming degrees occurred during controlled respiration in the other patients. All patients seemed to be hypersensitive to the inhibitory effects of neostigmine on the heart and required unusually large doses of atropine to counteract this effect.

For the subsequent twenty adult laparotomies the dose of d-tubocurarine was restricted to 5–10 mg, depending on the size of the patient. This dosage produced satisfactory relaxation with spontaneous respiration which required assisting in a few cases. Two mitral valvotomies were successfully performed with this technique, using only assisted respiration throughout; in both these cases the relaxation provided by the Fluothane (scale mark $6\frac{1}{2}$) was quite adequate, the d-tubocurarine being given to depress a little the spontaneous respiration. Neostigmine was considered necessary for a few cases in this series and severe bradycardia occurred in spite of a reasonable dose of atropine. In view of the consistent hypersensitivity to the cardio-inhibitory effects of neostigmine the further use of d-tubocurarine during Fluothane anaesthesia was abandoned.

Suxamethonium in 50 mg doses intravenously was then administered during Fluothane anaesthesia. It caused no further fall in systolic pressure, in fact the systolic pressure frequently increased by from 10 to 15 mm Hg. Similarly, controlled respiration during suxamethonium apnoea under Fluothane (scale mark 7) did not produce any obvious impairment of the peripheral circulation. Over 200 patients have now received suxamethonium 50 mg intravenously under Fluothane anaesthesia without any adverse effects on their cardiovascular systems. In the management of abdominal cases a small dose of suxamethonium (30 to 50 mg) is given intravenously as the peritoneum is incised so as to permit the easy exploration and exposure of the pathological viscus. Subsequent doses are seldom required until the closure of the peritoneum when the suxamethonium is repeated, the Fluothane providing adequate relaxation and spontaneous respiration for the major part of the operation.

Heart Disease and Fluothane Anaesthesia.

Seventeen patients with clinical and electrocardiographic evidence of myocardial disease secondary to coronary insufficiency—eight of them had angina of effort and six had previous histories of congestive heart failure—have been anaesthetized with Fluothane (scale mark $6\frac{1}{2}$) for periods up to two hours. The operations included prostatectomies, cholecystectomies, and hemicolectomies. Throughout the periods of anaesthesia there was no significant evidence of further myocardial damage and all the patients recovered uneventfully. One of these patients had a severe anginal attack on arrival at the operating theatre and the details of the case are as follows:

CASE 1. Male, 48 years. Muscular and well nourished. For transthoracic cardiac neurectomy for the relief of intractable angina of effort. The electrocardiogram showed inverted T waves in the six V leads, with erect T waves in the classical limb leads. No cyanosis, oedema, or arteriosclerosis. Lungs normal. Blood pressure 120/85. Premedicated with atropine 0.6 mg and pethidine 50 mg. A few minutes after arrival at the theatre the patient complained of severe precordial pain and dyspnoea. His pulse rate was 115 beats a minute and his blood pressure 120/80, and the electrocardiogram showed depression of the ST segment on lead 2 (fig. 3A). Anaesthesia was immediately induced with thiopentone 200 mg intravenously (without additional atropine) followed by suxamethonium 50 mg and orotracheal intubation; this caused no change in the electrocardiogram. Anaesthesia was then maintained with oxygen, nitrous oxide 50 per cent, and Fluothane (scale mark 7). Smooth spontaneous respiration returned in three minutes by which time the pulse rate had dropped to 88 beats a minute and was of much better volume, the systolic pressure had fallen to 70 mm Hg; the skin became warm, dry, and pink, and the ST segment returned to its normal isoelectric position (fig. 3D). Fifteen minutes later, just prior to the opening of the pleural cavity, 7.5 mg of d-tubocurarine were administered intravenously and respiration gently assisted to prevent the complete collapse of the left lung. For the next twenty minutes anaesthesia was maintained with Fluothane (scale mark 6½), the systolic pressure persisted at 70 mm Hg, the pulse rate gradually slowing to a steady 65 beats a minute, and the skin remaining warm and dry. Fluothane was then discontinued and within five minutes, although the pulse rate and blood pressure were gradually rising, the skin became clammy and cool. Fluothane (scale mark 6½) was started again and in a few minutes the skin became warm and dry, the blood pressure stabilized at 70 mm Hg systolic and the pulse rate at 65 beats a minute. The operation was successfully completed after 80 minutes anaesthesia. Methoxamine hydrochloride 5.0 mg was injected intravenously at the end of the operation. Fifteen minutes later the patient was awake and rational with a blood pressure of 130 mm Hg systolic and a pulse rate of 88 beats a minute. Recovery was uneventful.

Eleven patients with electrocardiographic evidence of hypertensive heart disease have been anaesthetized with Fluothane for periods up to 2½ hours without the development of any significant evidence of further myocardial damage. in these patients the systolic pressure did not fall below 100 mm Hg. The following case history illustrates one

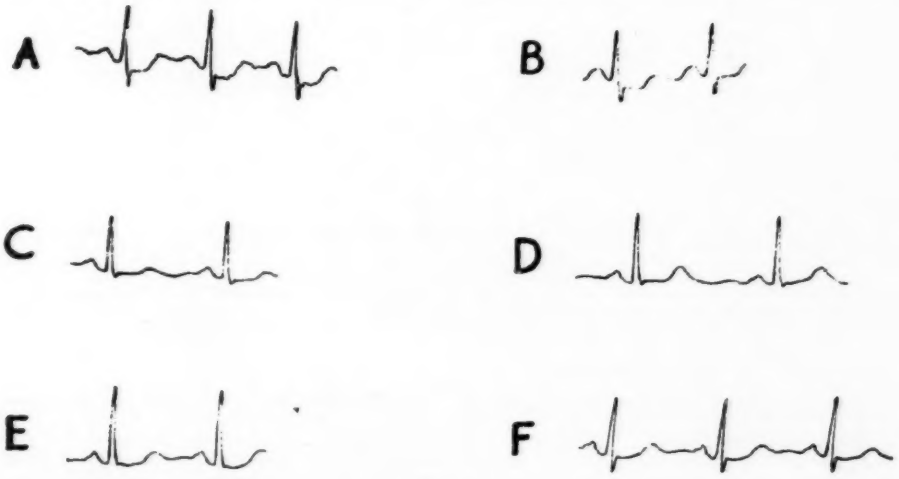

FIG. 3

Case 1. See text for clinical details. All Lead 2.

A. Immediately before induction.
B. After intubation under thiopentone and suxamethonium.
C. Three minutes later, under Fluothane anaesthesia.
D. After Fluothane anaesthesia for thirty minutes.
E. After Fluothane anaesthesia for sixty minutes, lighter plane.
F. Fifteen minutes after the end of the operation; patient conscious.

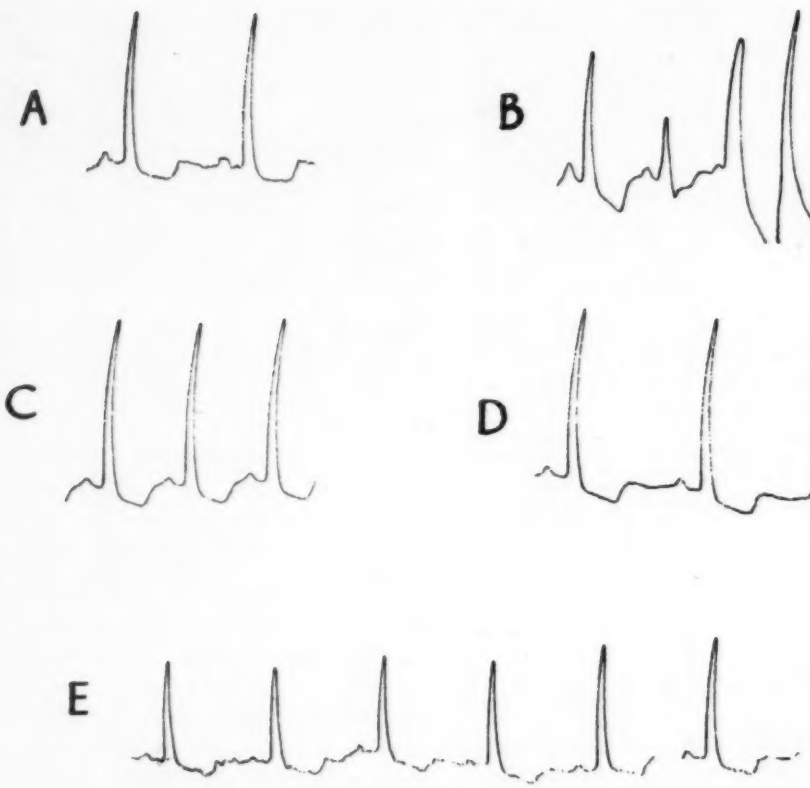

FIG. 4

Case 2. See text for clinical details. All Lead 1.

A. Before induction.
B. Immediately after induction with thiopentone; patient well oxygenated.
C. Twenty seconds later, after the insufflation of Fluothane vapour.
D. After Fluothane anaesthesia for two hours.
E. Twenty minutes after the end of the operation; patient conscious.

of the more severe hypertensives during Fluothane anaesthesia:

CASE 2. Female, 66 years. Craniotomy and removal of a right frontal glioma. Frail and thin. Fully conscious. Blood pressure 210/120 with electrocardiographic evidence of hypertensive heart disease (fig. 4A). No cyanosis or oedema, lungs normal. Premedication atropine 0.6 mg with pethidine 50 mg. Induced with thiopentone (without additional atropine) followed by suxamethonium 50 mg and orotracheal intubation. A severe derangement of cardiac function followed the injection of thiopentone (fig. 4B) and was abolished immediately (fig. 4C) by the rapid insufflation of Fluothane vapour (2 per cent approx. in oxygen); the patient was adequately oxygenated at the time of the appearance of this arrhythmia. Anaesthesia was then maintained for 2¼ hours with oxygen, nitrous oxide 50 per cent, and Fluothane (scale mark 6). During the period of anaesthesia the systolic blood pressure did not drop below 130 mm Hg and bleeding from the operation area was negligible. Blood replacement therapy was not necessary and the patient responded to instructions within fifteen minutes of the conclusion of the operation. Recovery was uneventful.

Effects of Fluothane on Cardiac Arrhythmias present before Induction.

Auricular fibrillation was present in eight patients before induction. The fibrillation was associated with coronary sclerosis in two patients, mitral stenosis in one patient, pulmonary emphysema in three, and thyrotoxicosis in two patients. Throughout the operations the auricular fibrillation was unchanged and the ventricular response decreased in each case. All patients made uneventful recoveries.

Frequent ventricular extrasystoles were observed in seven patients before operation. In six of these the ectopic beats disappeared completely during Fluothane anaesthesia but returned with the return of consciousness; one of these cases is illustrated in figure 5. The incidence of ectopic beats in the seventh case remained unchanged during the induction, maintenance, and recovery from anaesthesia.

Pulmonary Disease and Fluothane Anaesthesia.

Twenty-eight patients with chronic hypertrophic pulmonary emphysema have

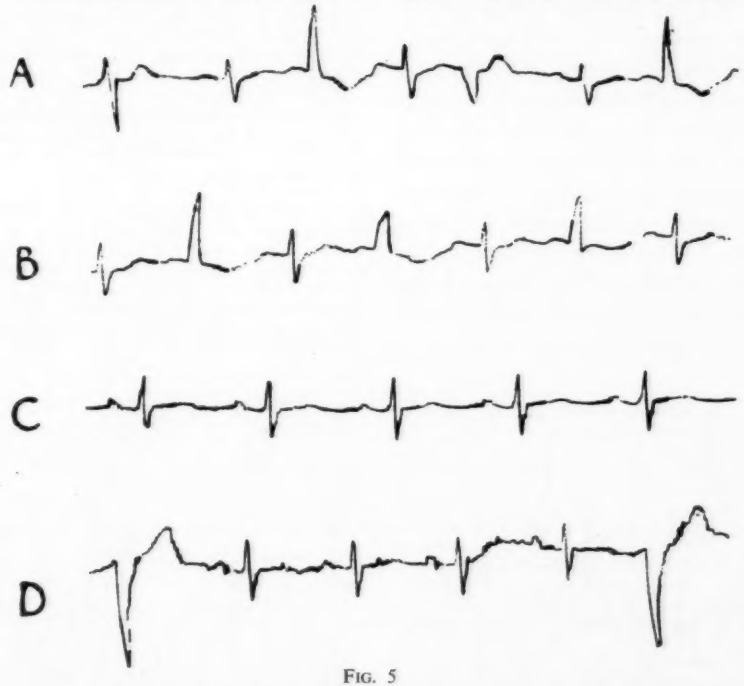

FIG. 5

Female, 75 years. For removal of a rectal polyp. Irregular pulse. No oedema or cyanosis. Blood pressure 140/90. Premedicated with atropine 0.6 mg with pethidine 50 mg. All Lead 2.

A. Before induction.
B. After thiopentone 200 mg intravenously.
C. After five minutes Fluothane anaesthesia.
D. During recovery of consciousness.

been anaesthetized with Fluothane. In one case the pulmonary disease was secondary to severe kyphoscoliosis, the remainder being due to chronic bronchitis. Three of these patients had severe cor pulmonale syndromes with orthopnoea, cyanosis, and congestive heart failure at intervals. The operations were mainly prostatectomies. Each patient was premedicated with atropine and pethidine and anaesthesia was induced with the usual sleep dose of thiopentone followed by suxamethonium 50 mg and the insertion of an airway; one patient was induced with Fluothane (scale mark 8) in oxygen. In each case the Fluothane anaesthesia was associated with smooth and easy respiration with the disappearance of the bronchiolar constriction associated with the disease; cyanosis disappeared and the patients became pink and well oxygenated; muscular relaxation was excellent and bleeding was minimal. Recovery of consciousness was prompt in all cases and there was no evidence of the shock syndrome. In the more severe cases there appeared to be an improvement in their respiratory functions postoperatively. Clinical details of one of these cases are as follows:

CASE 3. Male, 43 years. Haematuria and pyuria due to prostatic calculi. Severe kyphoscoliosis with cor pulmonale. Cyanosed, orthopnoeic, and confined to bed for the past six months; occasional oedema of the dependent parts. Blood pressure 130/90. Sinus rhythm, 125 beats a minute, with severe right ventricular strain.

First anaesthetic. Cystoscopy. Induced with thiopentone 250 mg intravenously followed by suxamethonium 50 mg and the insertion of an airway. Apnoea persisted for the next thirty minutes during which time the patient was ventilated with nitrous oxide in oxygen 30 per cent; during this period the pulse was rapid and feeble, the systolic blood pressure 70 mm Hg and sometimes much lower. A cyanotic pallor persisted throughout the period of anaesthesia and was associated with clammy sweating. Weak spontaneous respiration returned after thirty minutes and consciousness returned twenty-five minutes later. The patient was very distressed and dyspnoeic for several hours afterwards.

Second anaesthetic. Three months later, Fluothane now available. For suprapubic prostatectomy. Patient's cardiopulmonary condition unchanged. Patient arrived at the theatre in a severely orthopnoeic state, cyanosed and sweating, pulse rate 125 a minute, blood pressure 135/90; electrocardiograph showed sinus tachycardia with right ventricular strain (fig. 6A). Anaesthesia was induced with Fluothane (scale mark 8). After two minutes the patient was obviously unconscious but not adequately relaxed for easy intubation; 50 mg of suxamethonium were immediately injected intravenously and an orotracheal tube inserted without difficulty and without electrocardiographic disturbance. During the period of suxamethonium apnoea the

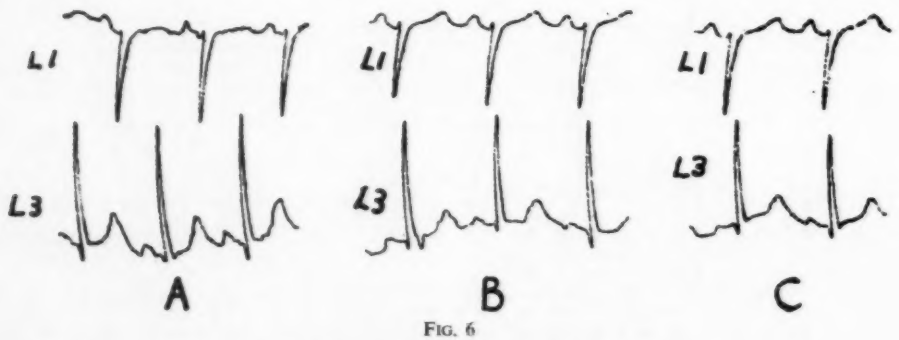

FIG. 6

Case 3. See text for details. Leads 1 and 3.

A. Before induction.
B. After Fluothane anaesthesia for forty minutes.
C. During the recovery of consciousness.

patient's lungs were very difficult to inflate but this difficulty disappeared with the return of spontaneous respiration three minutes later. For the next 90 minutes anaesthesia was maintained smoothly with Fluothane (scale mark 7). During this time respiration was spontaneous and offered no resistance to inflation. The systolic blood pressure was maintained at 70 mm Hg, with a regular sinus rhythm at 110 beats a minute (fig. 6B). Relaxation was excellent and bleeding was minimal—the operation was performed with the patient in the Trendelenburg position 30 degrees. Throughout the operation the patient's skin remained warm, dry, and pink. Consciousness returned within fifteen minutes of stopping the anaesthetic and recovery was uneventful. After the operation the patient stated that his breathing felt easier than it had done for several months.

In addition to the emphysematous patients there were three patients with suppurative bronchiectasis and three with active pulmonary tuberculosis. There were no operative or postoperative pulmonary complications in any of these patients. During anaesthesia with Fluothane there was a complete absence of salivary, mucous, and bronchial secretions in these patients.

Jaundice and Fluothane Anaesthesia.

Eight adult patients with jaundice have been anaesthesized with Fluothane for periods up to two hours. In seven patients the jaundice was due to carcinoma of the head of the pancreas and cholecystduodenostomy was done in each case; two of these patients had hypertensive heart disease, one with auricular fibrillation and intraventricular block. The eighth patient became jaundiced after an attack of acute cholecystitis. Each patient was premedicated with atropine 0.6 mg with pethidine 50 mg and anaesthesia was induced with the usual dose of thiopentone—with additional atropine—followed by suxamethonium 50 mg and orotracheal intubation. Anaesthesia was then maintained with Fluothane (scale mark 7). Maintenance of anaesthesia was smooth and uneventful in all cases, without any clinical or electrocardiographic evidence of further cardiovascular damage; recovery of consciousness was as quick as in the nonjaundiced cases. Seven patients made uneventful recoveries, the jaundice gradually disappearing in two or three weeks; one patient, with carcinomatous deposits in the liver, died on the seventh postoperative day from the effects of biliary peritonitis.

Uraemia and Fluothane Anaesthesia.

Fluothane (scale mark 6 to 7) was administered to ten patients with uraemia secondary to urinary obstruction due either to prostatic hypertrophy or to ureteric obstruction by pelvic neoplasm. In addition, twelve elderly males were admitted with uraemia secondary to prostatism; normal blood urea levels were restored in each of these cases by preoperative catheter drainage. Details of the patients who were still uraemic at the time of operation are as follows:

CASE 4. Female, 48 years. Anuria of at least 48 hours duration, secondary to obstruction of both ureters by pelvic neoplasm. Blood urea 380 mg per cent; serum potassium 6.54 mEq.; blood pressure 170/100 mm Hg. Patient drowsy and dehydrated, radial arteries difficult to palpate, suggesting vasospasm of considerable degree; no visible or palpable veins on arms or legs. Premedicated with atropine 0.6 mg and pethidine 50 mg.

Anaesthesia was induced with Fluothane (scale mark 8) and an orotracheal tube inserted without difficulty and without electrocardiographic changes after two minutes (fig. 7B). Anaesthesia then maintained for the next 45 minutes with Fluothane (scale mark 6½), during which time a left nephrostomy was performed without difficulty, relaxation being excellent, bleeding minimal, and spontaneous respiration remaining efficient.

Immediately after the induction of anaesthesia the systolic blood pressure dropped to 70 mm Hg and brisk vasodilatation occurred with the appearance of several large veins on the forearms which permitted the easy intravenous administration of fluids and glucose to combat the potassium intoxication. The

electrocardiogram, which pre-operatively showed evidence of potassium intoxication (fig. 6A), remained completely unchanged throughout the operation.

Consciousness returned five minutes after the cessation of Fluothane inhalations and the blood pressure returned to 170 mm Hg systolic a few minutes later. There was no postoperative nausea or vomiting. Immediately after the operation there was a steady flow of urine from the nephrostomy and three days later the blood potassium was subnormal (fig. 7); this was rectified by the oral administration of potassium. The blood urea, after a gradual rise to 440mg per cent on the third day after the operation, had returned to normal by the 21st postoperative day.

CASE 5. Female, 51 years. Anuria for two days as the result of ureteric obstruction by pelvic neoplasm. Fully conscious, condition good, blood urea 120 mg per cent, serum potassium normal. Induced and maintained with Fluothane for thirty minutes during which time urinary flow was re-established by the insertion of ureteric catheters. Blood chemistry gradually returned to normal in seven days.

CASE 6. Female, 53 years. Clinical details essentially similar to Case 5. After twenty minutes of Fluothane anaesthesia, a pre-operative blood urea of 153 mg per cent had returned to normal a week later.

CASE 7. Male, 75 years. Acute retention of urine due to prostatic hypertrophy. Completely disorientated and confused, blood urea 135 mg per cent. Heart and lungs normal. Anaesthesia induced with thiopentone 250 mg (with additional atropine) and maintained by Fluothane (scale mark 7) for 65 minutes whilst a suprapubic prostatectomy was successfully performed. Consciousness returned fifteen minutes after the end of the operation and there was no evidence of shock. Twenty-four hours later the patient was quite rational and the blood urea returned to normal seven days later.

CASE 8. Male, 83 years, with prostatic hypertrophy. Blood urea 243 mg per cent on admission and reduced to 87 mg per cent after four weeks catheter drainage. A suprapubic prostatectomy was successfully performed under Fluothane anaesthesia lasting 55 minutes. Postoperatively there was a progressive rise of the blood urea level to 350 mg per cent on the 15th day; this was associated with a diuresis and normal blood electrolytes most of the time. Seven days later the blood urea had returned to 83 mg per cent and the patient had become ambulant and active.

CASE 9. Male, 68 years, with prostatic hypertrophy. Blood urea on admission was 123 mg per cent and reduced to 74 mg per cent by seven days catheter drainage. Suprapubic prostatectomy then performed under Fluothane anaesthesia. Postoperatively the blood urea rose to 112 mg per cent on the third day and was within normal limits by the tenth day.

CASE 10. Male, 79 years. Clinical details essentially similar to Case 9.

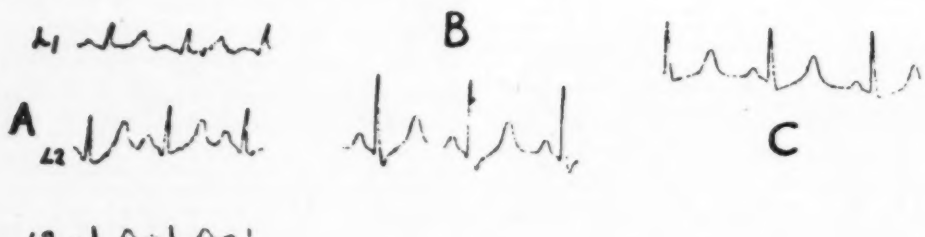

FIG. 7

Case 4. See text for clinical details. All, except A, are Lead 2.

A. Before induction, Leads 1, 2, and 3. Serum potassium 6.54 mEq.
B. After induction and intubation under Fluothane anaesthesia.
C. After forty minutes of Fluothane anaesthesia.
D. During the recovery of consciousness.
E. Three days later. Serum potassium 3.08 mEq.

CASE 11. Male, 67 years. Clinical details essentially similar to Case 9.

CASE 12. Male, 63 years. Clinical details essentially similar to Case 9.

CASE 13. Male, 68 years. Clinical details essentially similar to Case 9.

Deaths following Surgery under Fluothane Anaesthesia.

No deaths have occurred under Fluothane anaesthesia. Six patients have died in the postoperative period and the details are as follows:

(1) Female, 47 years. Bilateral adrenalectomy for generalized carcinomatosis due to malignant disease of the breast. Pathological fractures of the ribs, pleural effusions, and multiple secondaries in the liver. Emaciated and anaemic. The bilateral approach to the adrenals was used and the patient tolerated the operation very well. Blood pressure remained within normal limits throughout and consciousness returned five minutes after the completion of the operation. Twenty-four hours later the blood pressure began to fall and the patient died twenty hours later in spite of the intensive use of vasopressors of various kinds.

(2) Male, 61 years. Died seven days after a suprapubic prostatectomy. Autopsy revealed bilateral suppurative pyelonephritis and old standing suppurative bronchiectasis. Cause of death was toxaemia due to sepsis.

(3) Male, 68 years. Amentia secondary to cerebral atrophy as the result of cerebral arteriosclerosis. Bedridden and incapable for several months. Scattered moist sounds at both lung bases. Cerebral angiography and pneumoencephalography performed under Fluothane anaesthesia of 65 minutes duration. Consciousness and full reflexes returned within fifteen minutes without any evidence of shock or collapse. Prior to recovery of consciousness a considerable amount of purulent sputum was aspirated from the bronchi. The patient died 48 hours later from what appeared to be hypostatic pneumonia. Autopsy revealed a terminal bronchopneumonia.

(4) Baby, seven months old. For removal of a huge occipital meningoencephalocele. Heart and lungs normal. Premedicated with atropine 0.4 mg. Anaesthesia induced with Fluothane (scale mark 8). Intubation of the trachea was impossible because of the extreme degree of neck flexion caused by the tumour. A nasopharyngeal tube was inserted and anaesthesia smoothly maintained for the next 2½ hours with Fluothane (scale mark 7). During this time the meningocele was excised along with a large amount of brain tissue. Blood loss was moderately severe and was not satisfactorily replaced. The baby commenced to cry and move its limbs within five minutes of stopping the anaesthetic. Death occurred twelve hours later and permission for autopsy was not obtained.

(5) Male, 73 years. Cystoscopy and suprapubic prostatectomy. Fit and well nourished. Heart and lungs normal, blood pressure 170/100. Blood urea 83 mg per cent Twenty-six years history of diabetes mellitus, having insulin 20 units twice daily with appropriate diet. Blood sugar immediately before operation was 240 mg per cent. No urinary evidence of ketosis. Premedicated with atropine 0.6 mg and pethidine 50 mg. Anaesthesia induced with thiopentone 250 mg (with additional atropine 0.4 mg) followed by suxamethonium and the insertion of an airway. The operation was performed under Fluothane (scale mark 6½) without difficulty in 75 minutes, relaxation was adequate without relaxants, bleeding was minimal, and the systolic blood pressure did not drop below 110 mm Hg. Blood transfusion was not required. Recovery from anaesthesia was complete in fifteen minutes.

Twelve hours later the patient became restless and the blood sugar rose to 480 mg per cent. The appropriate therapy was instituted, but the response was unsatisfactory, and the patient died from what appeared to be peripheral circulatory failure 36 hours later. During the first 24 hours postoperatively there was a satisfactory urinary output. Hypokalaemia was not excluded. Autopsy failed to reveal the cause of death.

(6) Female, 41 years. Craniotomy and partial removal of a pituitary tumour. Obese. Heart and lungs normal. Premedicated with atropine and pethidine. Anaesthesia induced and maintained for 3½ hours with Fluothane. Consciousness returned within fifteen minutes after the end of the operation and the patient's condition was excellent for the next twenty-four hours. A sudden and unexpected collapse then occurred and the patient died within a few minutes. Autopsy failed to reveal the cause of death.

DISCUSSION

The general picture of the human cardiovascular system during Fluothane anaesthesia is one of vasodilatation combined with hypotension and bradycardia. The vasodilatation—as evidenced by dry, warm, and pink skin with prominent superficial veins—becomes obvious within a few seconds after the start of inhalations and persists throughout the period of anaesthesia, even when minimal concentrations of vapour are inhaled. It does not appear to be affected by haemorrhage or by surgical stimuli.

The hypotensive effect is directly related to the inhaled vapour concentration, being minimal with the lower concentra-

tions used for the maintenance of anaesthesia. In just less than half of the patients the hypotensive effect diminished as the anaesthetic continued: whether this is due to an increasing tolerance to the drug or to a diminishing output by the vaporizer cannot be stated but it is obvious that, for a drug of this potency, much more accurately controlled and calibrated vaporizers should be incorporated in the anaesthetic apparatus. Sudden haemorrhage causes an immediate and precipitous fall in systolic pressure, and the response to intravenous transfusion of blood or saline is equally rapid. The hypotensive effect of Fluothane can be safely prevented or abolished by the intravenous administration of methoxamine, if considered necessary. Patients under Fluothane anaesthesia are particularly sensitive to the vasopressor effects of methoxamine and doses in the region of 5 mg are usually adequate.

The use of d-tubocurarine with controlled respiration would appear to be contra-indicated during Fluothane anaesthesia. It would seem that d-tubocurarine, which is known to depress synaptic conduction in the sympathetic ganglia (Langley, 1918; Robbins and Lundy, 1947), in these circumstances causes such a profound degree of sympathetic block that intermittent positive pressure respiration obstructs the flow of blood through the lungs with consequent collapse of the systemic circulation.

Some degree of slowing of the pulse rate has always followed the administration of Fluothane, even to patients premedicated with atropine 1.0 mg. The mechanism of the slowing is not yet fully understood, but it is undoubtedly based on overactivity of the cardiac vagus. Whether this vagal overactivity is due to active stimulation of the parasympathetic nerves to the heart, or to an increase in vagal tone secondary to depression of the normal sympathetic counteraction, remains to be seen. Until further knowledge is obtained it would seem to be advisable to premedicate all patients with atropine in the appropriate dosage.

Light Fluothane anaesthesia has produced only minimal electrocardiographic evidence of increased irritability of the human heart. Twelve patients developed ventricular extrasystoles during the maintenance of light anaesthesia in circumstances in which the elimination of carbon dioxide was obviously impaired. Several other patients were subjected to similar degrees of ventilatory impairment and failed to show any electrocardiographic evidence of arrhythmia. Ventricular tachycardia has not been observed in any patient.

The most striking effect of Fluothane anaesthesia has been the complete absence of the shock syndrome—tachycardia, hypotension, sweating, pallor, and vasoconstriction—during and immediately following surgical operations. This was particularly evident after the more severe operations and especially in patients with advanced cardiovascular and respiratory diseases. Postoperative hypotension persisted for one or two hours in three patients in whom the replacement of blood loss was either inadequate or tardy; nevertheless, their skins remained warm and dry, their pulse rate averaged 90 beats a minute, and the recoveries of consciousness were not retarded.

The apparently complete protection which Fluothane anaesthesia has provided against surgical shock in this investigation may be related to a suppression of sympathetic activity during anaesthesia. Evidence is accumulating which indicates that blockade of the sympathetic division of the autonomic nervous system during surgery prevents the type of circulatory collapse which may lead to death (Martin, 1955; Overton and De Bakey, 1956; Ross and Herczeg, 1956; Hershey et al., 1956). The excellent results obtained with Fluothane anaesthesia in patients with severe pulmonary emphysema and cor pulmonale may also be related to its effects on sympathetic ganglia: Bromage (1956) has recently demonstrated that ganglionic blockade caused significant increases in the vital capacities of patients with pulmonary hypertension.

As the result of recent clinical observations it has been suggested that Fluothane anaesthesia may be followed by a diuresis which may cause hypokalaemia in those patients whose potassium levels are already diminished by disease. Although the evidence is by no means complete, it would be advisable to ensure adequate electrolytic and fluid replacements in those patients who present evidence of electrolytic depletion prior to or after anaesthesia with Fluothane.

One elective Caesarean section was successfully performed under Fluothane (scale mark $7\frac{1}{2}$) anaesthesia. Relaxation was adequate, bleeding minimal, the child was fully conscious when delivered from the uterus, and the uterus retracted normally after the removal of the placenta. Recovery of consciousness was complete in seven minutes after the removal of the face mask and there was no evidence of shock, vomiting, retching, or nausea.

SUMMARY

(1) Fluothane—a new nonexplosive volatile anaesthetic agent—has been administered to 500 patients, including many with cardiovascular, pulmonary, renal, and hepatic diseases complicating the surgical lesions. Each patient was premedicated with atropine, either alone or combined with pethidine, and anaesthesia was induced in most cases with a sleep dose of thiopentone.

(2) Smooth and rapidly reversible anaesthesia has been maintained in all cases by the continuous administration of Fluothane vapour, using a Boyle's vaporizer and a gas flow of ten litres a minute with 50 per cent oxygen and nitrous oxide.

(3) Cardiovascular changes suggestive of depression of sympathetic activity have consistently been observed and which, to a certain extent, were modified by atropine and by the strength of the vapour concentration inhaled. The shock syndrome has been completely absent in all cases.

(4) There has been a complete absence of salivary, mucous, and bronchial secretions in all cases throughout the period of anaesthesia. Nausea, vomiting, and retching during recovery have been absent in over 90 per cent of the patients.

(5) Cardiac arrhythmias of the vagal type were observed when the higher concentrations of Fluothane vapour were inhaled. These arrhythmias included sinus bradycardia, atrioventricular nodal rhythms often with interference, dissociation, and an unusual form of sinus

arrhythmia with coupling of the sinus beats. Atropine restored normal sinus rhythms in these cases. Their incidence has been completely eliminated by adequate atropinization.

(6) Ventricular arrhythmias have been observed only in the presence of inadequate ventilation in lightly anaesthetized subjects and were of a mild degree. Ventricular tachycardia has not been observed.

(7) Cardiovascular collapse—slow pulse and profound hypotension—has followed the combined use of d-tubocurarine and controlled respiration on patients anaesthetized with Fluothane. Such reactions did not occur when suxamethonium and controlled respiration were used in conjunction with Fluothane anaesthesia.

(8) Tachypnoea has occurred during surgical stimulation in patients lightly anaesthetized with Fluothane. The reaction can be controlled by pethidine or by regional nerve block.

(9) No deaths have occurred under anaesthesia. The details of six postoperative deaths have been presented.

(10) It has been suggested that Fluothane anaesthesia may cause an immediate postoperative diuresis which may lead to serious electrolytic depletion in those patients already in a state of electrolytic deficiency as the result of disease.

ACKNOWLEDGMENTS

I should like to express my sincere thanks to Dr. Suckling of the General Chemicals Division of Imperial Chemical Industries Ltd., Widnes, for providing supplies of Fluothane: and also to Dr. Raventos and Dr. Paget of Imperial Chemical (Pharmaceuticals) Ltd., Manchester, for their constant assistance. I am also most grateful to my surgical colleagues at the Royal Infirmary and at Crumpsall Hospital, Manchester, for their co-operation.

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The Yorkshire Society of Anaesthetists are hoping to hold a three-day meeting in Copenhagen, starting on June 2, 1957. Any anaesthetist who would like to join them would be very welcome and preliminary information may be obtained from:

DR. GWENDOLEN HARRISON, 8 Shire Oak Road, Leeds, 6.

Brit. J. Anaesth. (1956), **28**, 411

PHYSIOLOGICAL MEASUREMENTS DURING ANAESTHESIA WITH FLUOROMAR

BY

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TRIFLUORO-ETHYL-VINYL-ETHER (Fluoromar) has been reported (Orth, 1955) to be a non-irritating, rapid-acting anaesthetic agent. The lower flammability limit is 4 per cent in oxygen or air (ethyl ether, 2 per cent) and the minimum ignition energy is higher by many fold than that of diethyl ether or divinyl ether at the same concentrations (Moen, 1956). It is obviously desirable to minimize the explosive hazard in operating rooms, so it seemed worth while to investigate some of the variations in physiological functions that might occur during the use of Fluoromar as an anaesthetic agent.

METHODS

Preliminary observations were carried out using dogs as subjects. They were anaesthetized with Fluoromar either (*a*) with a mask directly, (*b*) after introduction of an endotracheal tube following administration of a paralytic dose of suxamethonium, or (*c*) after a small intravenous dose of thiopentone. The animals were allowed to awaken from the thiopentone anaesthesia and then anaesthetized with Fluoromar through the endotracheal tube. Adrenaline was given to 4 of these animals, both before and after anaesthetization with Fluoromar, according to the technique of Meek, Hathaway and Orth (1937).

Surgical patients were anaesthetized by three different techniques. (1) Open drop technique was used with infants and children. (2) Anaesthesia was induced in patients with thiopentone and maintained using a closed circle absorption system with Fluoromar as the anaesthetic agent throughout the rest of the procedures. (3) Twelve patients were studied using Fluoromar as the only anaesthetic agent. Control chemical analyses were required, so these patients were selected on the basis of their being in the hospital at least two days pre-operatively in order that bleeding (Duke, 1919) and coagulation (Lee and White, 1913) times, blood volumes (Crispell, Porter and Nieset, 1950; Storaasli, Kreiger, Friedell and Holden, 1950), blood glucose (Nelson, 1944) and blood urea (Karr, 1924), as well as bromsulphalein (BSP) retention tests (Rosenthal and White, 1925) and urea clearances (Karr, 1924; Folin and Youngberg, 1919; Youngberg, 1921) could be done before surgery. Blood glucose and blood urea were determined on a sample taken immediately before in-

duction. Anaesthesia was induced with Fluoromar and oxygen using a closed circle absorption system. Tracheal intubation was performed. No anaesthetic or relaxing agent other than Fluoromar was used. After one hour of anaesthesia, the bromsulphalein retention, blood volume, bleeding and coagulation time, blood glucose and blood urea values were again determined. Fluoromar was therefore given to the patient for somewhat more than 1.5 hours before the surgery was started BSP retention was followed for several days or until it had returned to nearly control values. Urea clearance and blood glucose determinations were repeated the morning following surgery.

During these anaesthetic procedures the Monaghan ventilation meter was used to determine the effect of Fluoromar on tidal volume. Electroencephalographic tracings were made. The surgical procedures carried out using this technique included: 1 cholecystectomy; 1 laminectomy; 3 hysterectomies; 2 radical breast excisions; 1 breast biopsy; 1 excision of ovarian tumour; 1 pelvic node dissection; 1 femoral arterial graft; 1 gastrectomy. Electrocardiographic tracings were made at frequent intervals throughout 10 of these procedures.

RESULTS

The results may be divided into three parts; one includes the work with dogs; the second describes the measured values from the 12 patients in group 3; the third involves clinical observations.

I. *Results Using Dogs.*

Preliminary work with dogs indicated that Fluoromar was not especially irritating, that respiration was slightly if at all affected, and that salivation was minimal.

Control injections of adrenaline to 4 animals produced ventricular abnormalities as would have been expected. After the animals were asleep with Fluoromar, adrenaline injections were repeated with at least 15 minutes equilibration on Fluoromar and at least 30 minutes between injections of adrenaline. The records showed fewer irregularities when adrenaline was injected during the Fluoromar anaesthesia than during the control period. After waking, one of the animals was anaesthetized for 15 minutes with cyclopropane and then adrenaline injected a third time, allowing more than 30 minutes between adrenaline injections. This dog developed ventricular fibrillation (see chart 1).

II. *Laboratory Determinations.*

Table I* presents the analytical values obtained from the 12 patients in group 3. No significant changes in the values of the coagulation time, total blood volume, red cell or plasma volume, haematocrit or blood urea were found during the course of these observations. Bleeding time increased from an average 97 seconds to 123 seconds following one hour of anaesthesia with Fluoromar. These values had returned to

*See page 421.

CHART 1 (*opposite*)

7 kilogram dog.

A. Control awake, 10.55 a.m.
B. Adrenaline, awake, 11.00 a.m.
C. Asleep with Fluoromar, 12.06 p.m.
D. Adrenaline while asleep with Fluoromar, 12.08 p.m.
E. Asleep with cyclopropane, 12.35 p.m.
F. Adrenaline while asleep with cyclopropane, 12.42 p.m.

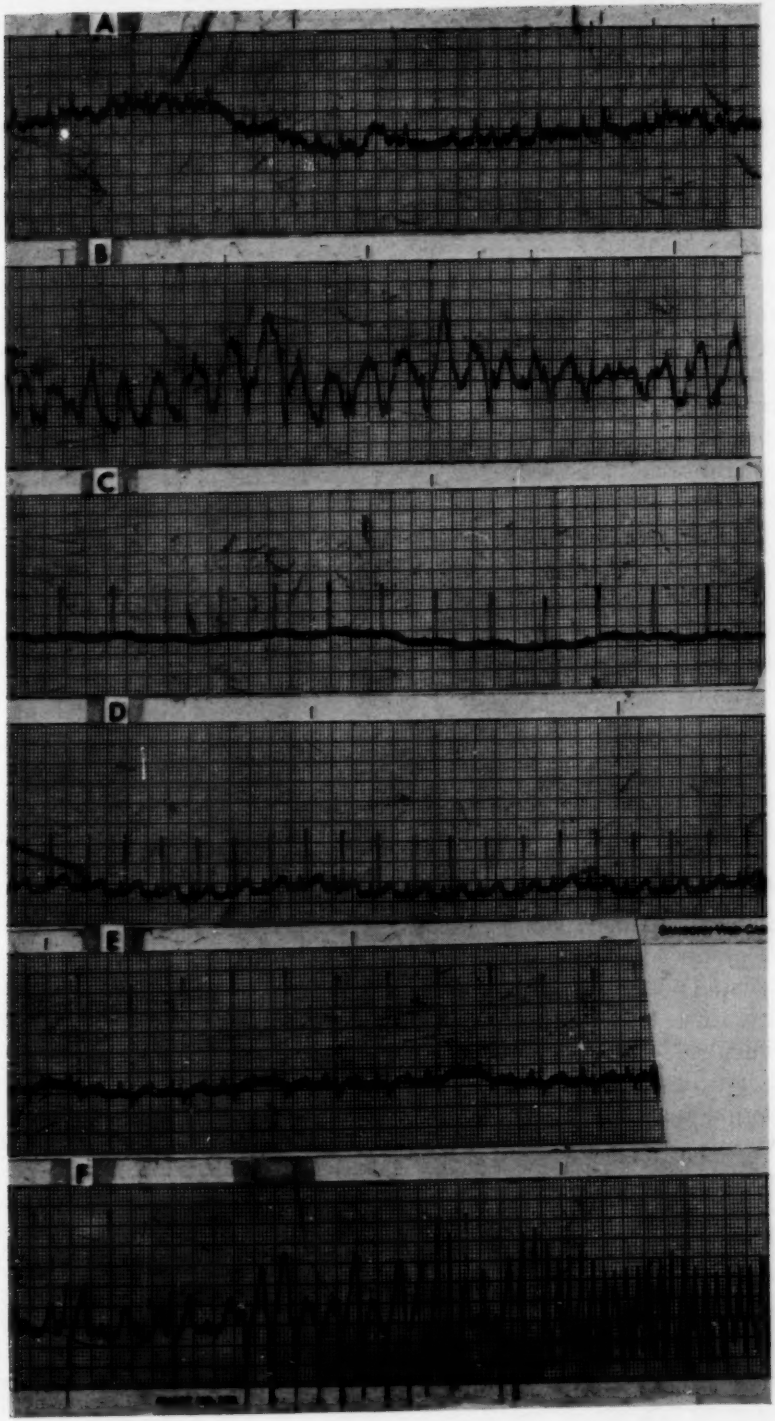

CHART 1

normal (94 seconds) the first postoperative day. Blood glucose values were significantly elevated from an average of 80 to 114 mg/ml during an hour of Fluoromar anaesthesia. BSP retention was elevated at the end of the hour of Fluoromar anaesthesia from 7 to 21 per cent. Following operation, these BSP values returned to normal as rapidly as would have been expected after the use of other anaesthetic agents (Waters, 1951). Urea clearance tests the day following surgery showed normal values. Numerical values for these tests are not given because some of the clearance values were maximum clearance and some were standard clearance, and not enough were done to treat them statistically. However, all were within normal limits.

III. *Clinical.*

Observations have been made on 100 patients. Fluoromar was not an irritating agent. The patients who received it were not excited by breathing this material. In fact, 3 of the 12 patients who were the subjects of group 3 requested specifically that they should not have an ether anaesthetic, but went to sleep with Fluoromar with no difficulty.

Patients lost consciousness rapidly when induced with Fluoromar. The induction, however, was not as rapid as it would have been with cyclopropane. They tolerated induction well with the Heidbrink absorber set at 6. There was no laryngospasm and no remarkable excitement period. Fluoromar seemed to prevent excessive "Bucking" on tracheal tubes. A few patients "bucked" when anaesthesia was quite light but, generally speaking, intubation was not stimulating to the patients. From 10 to 25 minutes were required before intubation could be carried out with Fluoromar as the sole agent administered. However, it was observed that when the patient's jaw could be opened, the cords were abducted. Fluoromar caused no excessive salivation. patients who had not received a belladonna drug had appreciable laryngeal secretions but seemed to salivate much less than would have been expected had ether been the anaesthetic agent. Ventilation by the patient was quite adequate so long as anaesthesia was light. Deeper planes of anaesthesia caused reduction in tidal volume. The physiological effects of Fluoromar differ from those of the commonly used anaesthetic agents, so the planes and stages of anaesthesia with Fluoromar have not been easy to determine by the usual criteria. Fluoromar had one respiratory effect similar to that of cyclopropane in that following a few large forced breaths, apnoea easily ensued. Assisted respiration very easily became controlled respiration.

Hypotension occurred in several patients without warning. It appeared before the usual signs of deep anaesthesia were noted and occasionally occurred while the patient was still moving.

Electroencephalography with these patients produced the same types of tracings as have been observed with ether (Courtin, Bickford and Faulconer, 1950). They were not consistent with the usual clinical signs of depth of anaesthesia. Hypotension occurred without warning of change of state on the electroencephalogram. Reliance on e.e.g. patterns for

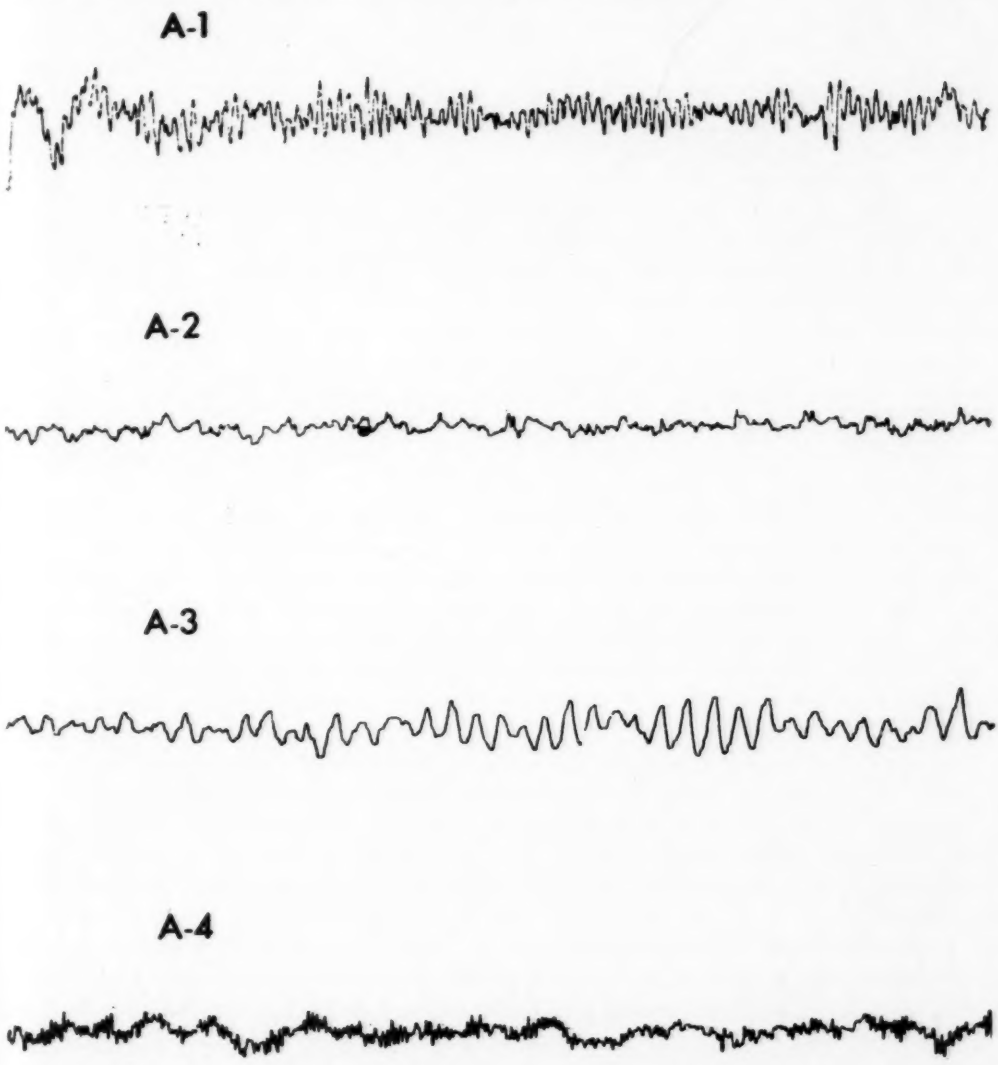

FIG. A
E.E.G. tracings

A—1. Awake. Systolic pressure 130 mm Hg.
A—2. Light anaesthesia. Systolic pressure 130 mm Hg.
A—3. Light anaesthesia. Systolic pressure 40 mm Hg.
A—4. Third plane anaesthesia. Blood pressure 50/20 mm Hg.

control of administration of Fluoromar seemed unjustified because severe hypotension frequently preceded any e.e.g. change. A few electroencephalographic patterns are presented. Figure A-1 is that of a patient awake whose systolic blood pressure was 130. Figure A-2 is the same patient in light clinical anaesthesia with blood pressure unchanged. Figure A-3 shows a record from the same patient who was still in light clinical anaesthesia but whose systolic blood pressure had dropped to 40 mm Hg due to the use of Fluoromar.

Figure A-4 shows the same patient in what was judged to be plane 2 of the third stage of anaesthesia clinically but whose blood pressure was 50/20 mm Hg. The low blood pressures in A-3 and A-4 followed immediately the use of high concentrations of Fluoromar. The pressures in both cases rose to normal within two minutes of eliminating Fluoromar from the breathing bag.

Figure B shows two records of a patient whose blood pressure dropped (due to Fluoromar) from 125 to 85 mm Hg. No clinical change was observed during this period. The e.e.g. voltage diminished moderately.

Figures C-1 and C-2 are e.e.g. records of a patient awake with a blood pressure of 114/75 mm Hg and asleep with a blood pressure of 89/50 mm Hg. When awake the tidal volume was 350 ml, when asleep the tidal volume was 450 ml. The second record was made when the patient was in light clinical anaesthesia.

Figure E is an e.e.g. made during Fluoromar anaesthesia which corresponds to the type that characterizes moderate surgical anaesthesia with ether. The blood pressure in this case was 90/70 mm Hg. The pre-induction blood pressure had been 110/70 mm Hg. Figure D is a record of a patient made when his blood pressure was 50 mm Hg systolic. This record is of the type corresponding to that showing deep anaesthesia when either cyclopropane or ether is used or a "danger" level with thiopentone. This patient was the only one of the series shown who was clinically deep in anaesthesia, that is, the pupils were dilating, fixed and dry; the tidal volume was only 50 ml and respiration was diaphragmatic in character. Pre-induction blood pressure was 100/60 mm Hg.

One striking feature observed during the use of Fluoromar was that when the blood pressure diminished, the pulse frequently slowed as well. The anaesthesia record (fig. F) is characteristic. No noticeable vasodilation occurred. Recovery has been rapid and without excitement.

Quite large quantities of this agent seemed necessary when using it by the open drop technique. By closed circle technique, the adult patients could be maintained in satisfactory surgical anaesthesia with approximately one ounce per hour of the material.

Some of the patients vomited. The list of operations indicates that surgery involved manipulation of the intestines so

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CHART 2 (*opposite*)

M.B., Cholecystectomy, 56 years, female. E.C.G. tracings.

A. Control awake, 07.35 a.m., BP 120/80 mm Hg.
B. Asleep with Fluoromar, 07.52 a.m., BP 100/80 mm Hg. Note—Transient loss of P wave.
C. Asleep with Fluoromar, 08.33 a.m. BP 105/85 mm Hg. Note—Transient P wave change.
D. Asleep with Fluoromar, 10.00 a.m., BP 110/85 mm Hg. Typical throughout procedure except for the two brief periods illustrated in B and C.

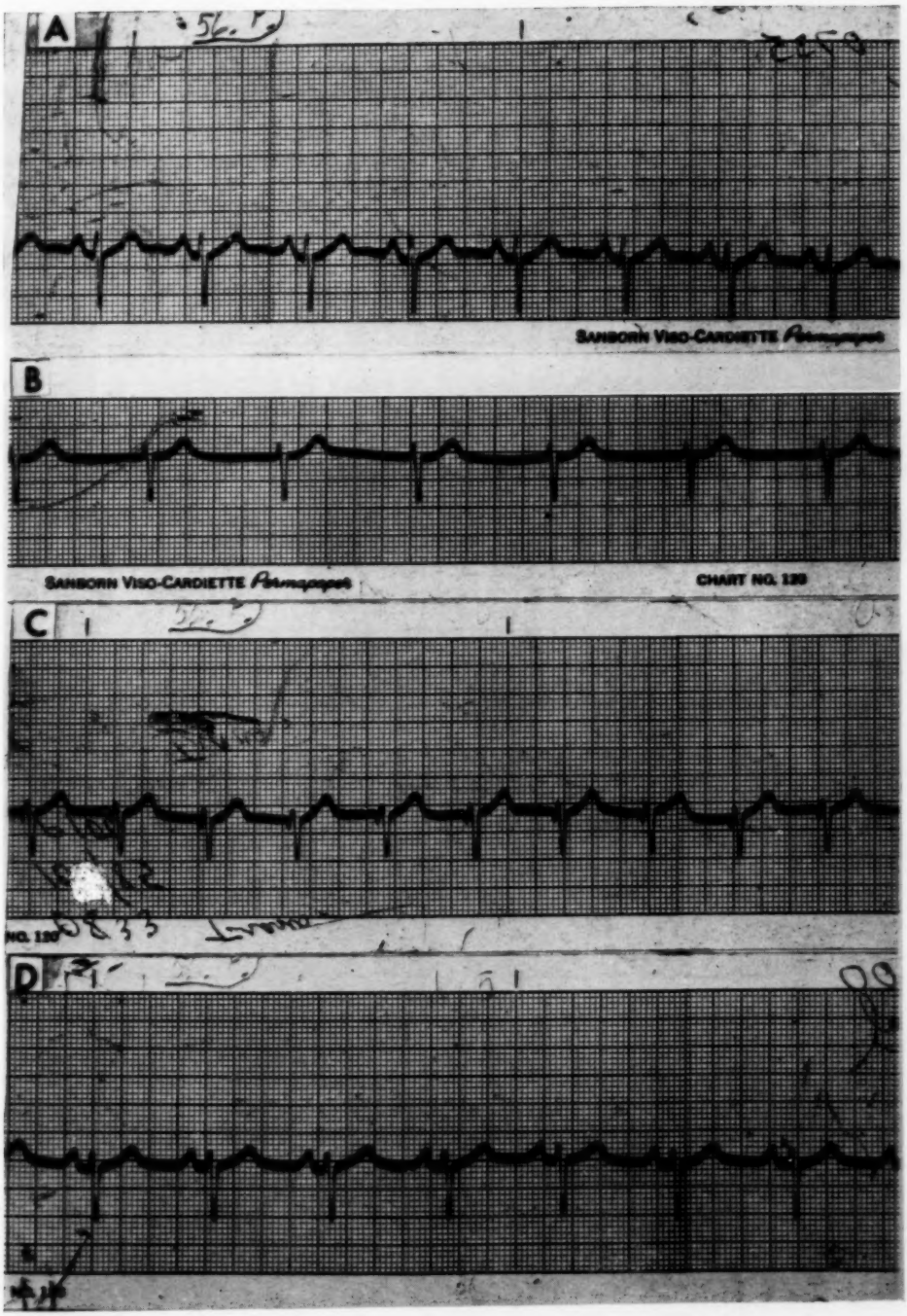
A
SANBORN VISO-CARDIETTE
B
SANBORN VISO-CARDIETTE
CHART NO. 120
C
D

CHART 2

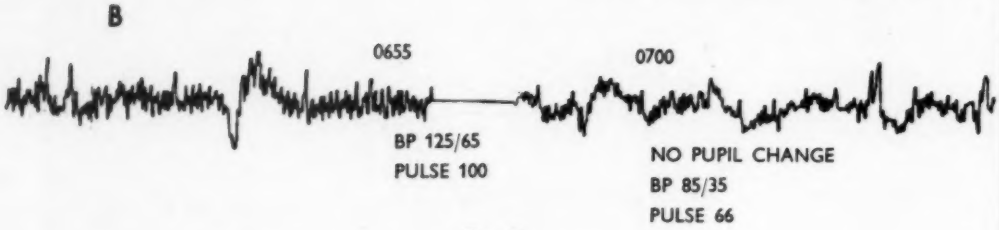

FIG. B
E.E.G. Tracings. Note clinical change. Note blood pressure change.

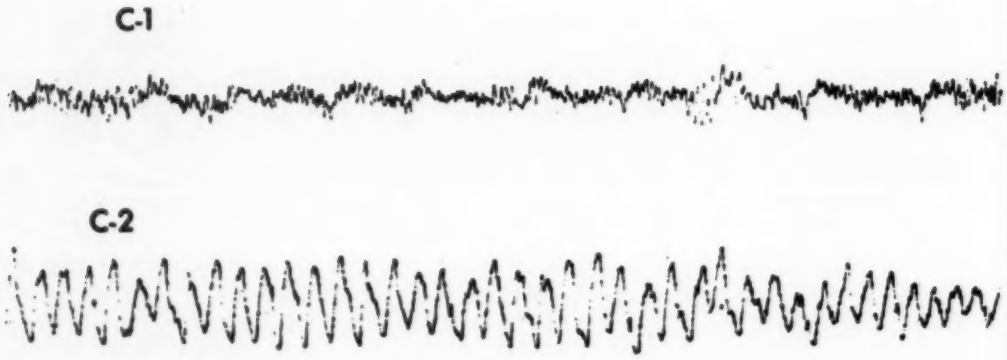

FIG. C
E.E.G. Tracings.

C—1. Patient awake. Blood pressure 114/75 mm Hg. Tidal volume 350 ml.
C—2. Patient asleep. Blood pressure 80/50 mm Hg. Tidal volume 450 ml.

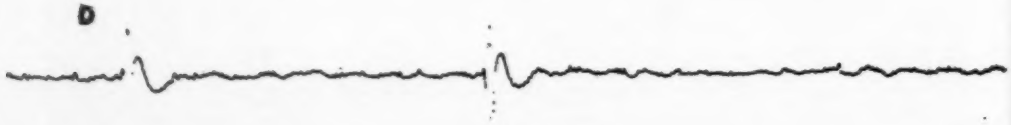

FIG. D
E.G.G. record. Plane 3, blood pressure 50 mm Hg systolic.

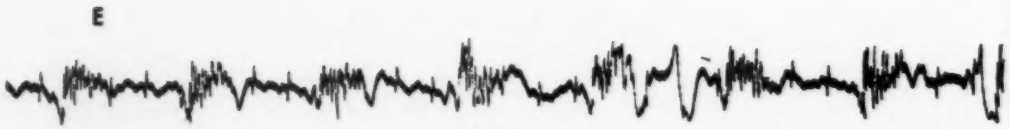

FIG. E
E.G.G. record. Plane 2, blood pressure 90/70 mm Hg.

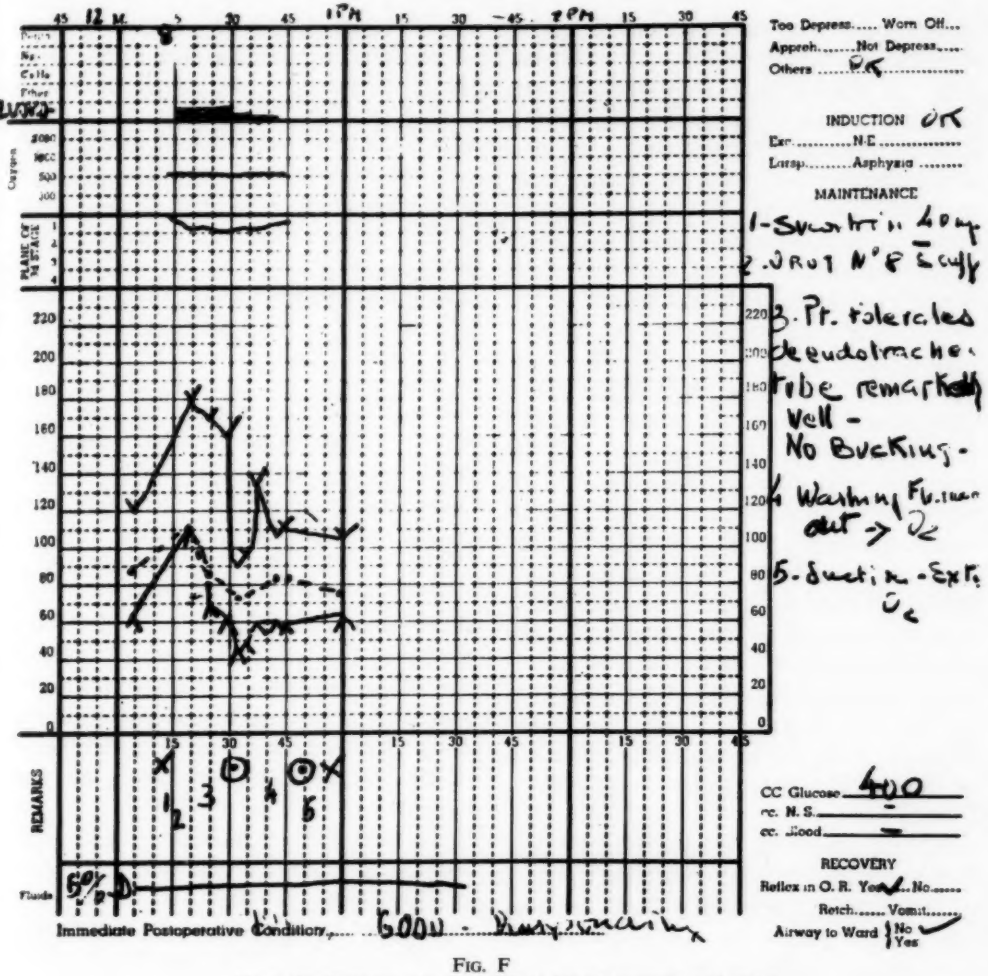

FIG. F
Note simultaneous rise and fall in blood pressure and pulse.

the vomiting cannot be attributed to this agent. The general incidence of vomiting after the use of this agent has been low.

Chart 2 shows the only type of electrocardiographic abnormality observed with human beings asleep with Fluoromar in this study. It consisted of a wandering pacemaker (change in P wave), which is not considered to be significant.

DISCUSSION

The administration of adrenaline to animals anaesthetized with Fluoromar was followed by very little electrocardiographic change. In all 4 animals in which this was done, the tracing under those conditions showed much less abnormality than when adrenaline was administered to the animals when they were awake.

Fluoromar therefore showed a "protective" effect on the myocardium of these dogs. It seems particularly significant that on administration of adrenaline during Fluoromar anaesthesia one dog showed only an increase in cardiac rate with no change in the contour of the curve, while that same dog had ventricular fibrillation when adrenaline was injected during cyclopropane anaesthesia.

The BSP retention test showed no significant deleterious effects of Fluoromar anaesthesia on the liver. It is realized that this measures only one phase of liver function. Waters (1951) measured BSP retention in a series of patients who were receiving chloroform and another series receiving other agents as controls. He observed that 25 per cent of those anaesthetized with agents other than chloroform showed abnormal dye retention. He also observed that when retention occurred, it disappeared within a period of 7 to 10 days. Our series shows this same parallelism for the values had returned to approximately pre-operative values within 7 days.

As measured by urea clearance and blood urea values, no deleterious effect of Fluoromar on kidney function was observed.

Following ether or chloroform anaesthesia, blood sugar would be expected to rise 100 to 200 per cent (Adriani, 1952). The increase of 34 mg per cent of blood glucose in these patients is comparable to what one would expect using cyclopropane as the anaesthetic agent rather than either ether or chloroform.

The effect on respiration was minimal in light planes of anaesthesia. In deeper planes, respiratory volume diminshed.

The effect on blood pressure seemed particularly significant. The mechanism of action is not known at this time so it cannot be stated whether use of Fluoromar is akin to controlled hypotension or not. Pressures returned to normal rapidly on discontinuance of the agent. Since cardiac output and tissue oxygenation in these circumstances are not known, no categorical statement can be made as to whether or not these hypotensive effects were good or bad. It would be desirable to determine cardiac output in these circumstances. Until more is known about the circulatory system during periods of hypotension it seemed unwise to rely on electroencephalographic patterns as a measure of depth of anaesthesia.

The lack of "bucking" with the use of Fluoromar seemed quite desirable. Certainly minute-to-minute control of the patient's condition is possible but it should be emphasized that such minute-to-minute or even second-to-second control is essential for its safe use. Inhalation of the material was pleasant for the patients.

The observations indicated that intubation can be done using Fluoromar alone but the time involved was such that routine use of this agent would probably be more efficient if the patient were induced with something like thiopentone and intubated using a relaxing agent before using Fluoromar alone.

The "wandering pacemaker" observed on the electrocardiographic tracing during Fluoromar anaesthesia has been observed with other inhalation agents (Kurtz, Bennett and Shapiro, 1936; Virtue and Pierce, 1951). Its exact significance has not been determined and certainly does

not point to a deleterious effect of Fluoromar on the myocardium.

SUMMARY

(1) Fluoromar is a nonirritating anaesthetic agent which produces little effect on respiration during early stages of anaesthesia.

(2) The common signs and stages of ether anaesthesia cannot be utilized for controlling administration of Fluoromar as an anaesthetic agent.

(3) Fluoromar frequently caused hypotension when no more than light planes of anaesthesia as judged by either e.e.g. tracings or the usual clinical signs of anaesthesia had been attained.

(4) Some increase in blood glucose, bleeding time and BSP retention was observed after one hour of Fluoromar anaesthesia.

(5) No significant change was observed in values of blood urea, total or fractional blood volumes, or coagulation time after one hour of Fluoromar anaesthesia.

(6) Kidney function was not measurably changed during one hour of Fluoromar anaesthesia.

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TABLE I

Physiological variations following the use of Fluoromar.

	No. pts.	Day before surgery		Pre-anaesthetic control		After 1 hr. of anaesthesia		1st post-operative day		Significant difference	
		Avg.	Std. dev.	Avg.	Std. dev.	Avg.	Std. dev.	Avg.	Std. dev.		
		A		B		C		D		Columns	Critical ratio
Bleeding time (secs.)	11	97.3	10.8			123.4	19.3	93.8	16.9	A–C	3.52
										C–D	3.46
Coagulation time (minutes)	12	13.8	5.6			11.3	5.0	14.8	6.0	None	
Total blood volume (ml)	10	4148	447			4143	863			None	
Plasma volume (ml)	10	2429	255			2434	470			None	
R.B.C. volume (ml)	10	1766	222			1737	529			None	
Haematocrit (%)	10	42.1	2.8			40.7	4.7			None	
Blood glucose (mg%)	10	81.8	10.8	80	5.9	114	22.8	91	20.4	A–C	3.91
										B–C	4.41
Blood urea (mg %)	12	13.5	3.7	13.1	4.5	13.8	4.5	11.8	3.8	None	
B.S.P. Retention (%)	11	8.6	7.5			21.2	7.9	*12.5	9.2	A–C	3.42

* B.S.P. values obtained 1 to 7 days later.

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PULMONARY VENTILATION AND CARBON DIOXIDE LEVELS DURING ANAESTHESIA

BY

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PULMONARY ventilation may vary widely during anaesthesia. Both anaesthetic agents and premedicant drugs (Eckenhoff et al., 1955) depress the respiratory centre; this central depression varies with the depth of anaesthesia and the dosage of the drugs used. During light anaesthesia surgical stimuli may "drive" the respiratory centre so that ventilation is temporarily increased (frequently such responses take the form of an increase in rate with little net effect on alveolar ventilation). Muscle relaxants impair ventilation by paralysis of the respiratory muscles; this paralysis may be complete and reduce pulmonary ventilation to zero. Operations involving opening of the chest also make it impossible for adequate respiration to be maintained spontaneously by the patient. For these reasons artificial respiration by the anaesthetist has become obligatory during a high proportion of anaesthetics.

The carbon dioxide levels of the patient may rise during anaesthesia as a result of two factors: (1) carbon dioxide accumulation in the apparatus (Bracken, 1956); and (2) hypoventilation, resulting from the causes considered above. Carbon dioxide depletion may occur when hyperventilation is produced by the anaesthetist and adverse effects have been described (Seevers et al., 1939; Brown, 1953), but it is generally agreed that the results are less serious than those of carbon dioxide retention.

It has been suggested that the ventilatory requirements can be assessed from physiological calculations based on the body weight of the patient and that such estimates, referred to as the "standard ventilation", be used as a guide to artificial respiration during anaesthesia (Scurr, 1956).

Observations of ventilation levels have been made in a series of patients anaesthetized by various techniques in order to determine the range of ventilation encountered in practice. The ventilation levels observed have been related to the physiological ventilation requirement derived from the standard nomogram (Radford, 1955).

STUDIES IN VENTILATION

The apparatus used is shown in fig. 1. An M.I.E. circle absorption apparatus was modified so that fresh gases entered at the angled bag mount, as shown. A dry gas meter (Parkinson and Cowan, Type D. 1) was placed across the inspiratory hose so as to measure all inspired gas volumes. A B.O.C. colorimetric carbon dioxide analyser was incorporated on the inspiratory side of the absorber to check that the

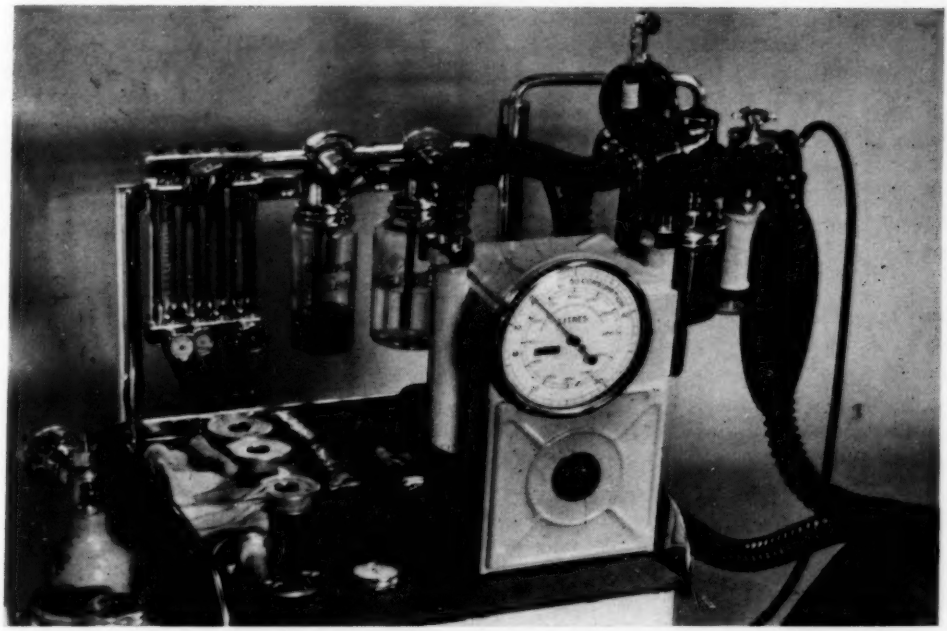

FIG. 1

inspired gases were free of carbon dioxide. The expiratory valve was on the further side of the meter from the patient. The inspired volume and respiratory rate were recorded every five minutes, as indicated by the stopwatch; the average minute volume for the period was then computed. For each period the standard ventilation was derived from the nomogram according to the body weight and the respiratory rate.

All patients were intubated and a gas-tight fit ensured by means of a cuffed tube or an efficient pack. The first chart (fig. 2) shows observed minute volumes expressed as a percentage of the physiologically calculated requirement, the abscissa indicating the duration of the anaesthetic. Observations made on eleven patients have been superimposed. These were all undergoing dental operations and the anaesthetic technique was standardized as follows: Premedication, h.i. papaveretum 20 mg and hyoscine 0.4 mg one hour pre-operatively. Induction, i.v. thiopentone 500 mg, gallamine 80 mg, pethidine 50 mg. Maintenance, nitrous oxide 5 litres per minute, oxygen 2 litres per minute. Unassisted spontaneous ventilation was allowed in all cases and the facial colour was satisfactory at all times. As might be expected, depression is maximal initially; after this early period respiratory volumes appeared adequate to ordinary clinical observation, but it is noteworthy that by physiological standards respiration was inadequate throughout in all patients. Records of respiratory

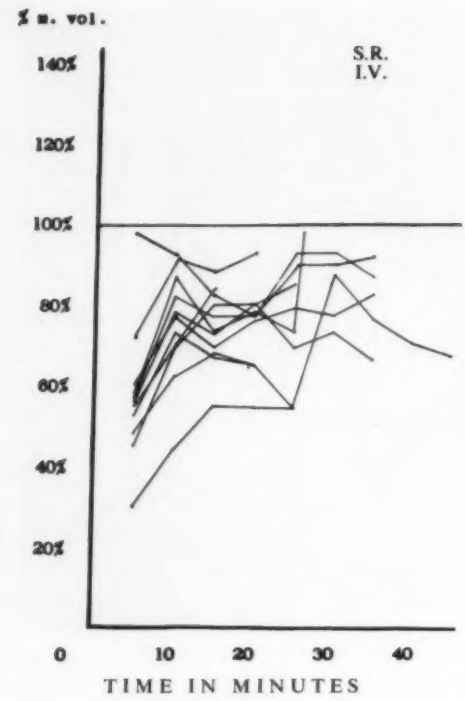

FIG. 2

Ordinate:
Observed minute volume expressed as percentage of standard.

Eleven patients:
Thiopentone, gallamine, pethidine, N_2O+O_2 (5:2)

depression and recovery during thiopentone anaesthesia in animals have been reported (Ament et al., 1949).

Cyclopropane is reputedly the most powerful respiratory depressant of inhalation anaesthetics. Figure 3 shows the percentage of standard ventilation observed in four patients anaesthetized to moderate depth with cyclopropane, following an induction with i.v. thiopentone 250 mg and premedication h.i. papaveretum 20 mg and hyoscine 0.4 mg. It is evident that respiratory depression occurs as cyclopropane anaesthesia is induced. Similar records obtained during ether anaesthesia following a thiopentone induction, perhaps surprisingly, showed a similar reduction in ventilation.

The "lytic cocktail" has a reputation for not producing severe respiratory depression, and, in keeping with this, Harbord and Eastwood (1955) reported relatively low carbon dioxide levels. Observations were made in eight patients, who received i.v. pethidine 100 mg, chlorpromazine 50 mg and promethazine 50 mg, following an induction dose of 200 mg thiopentone and premedication with papaveretum 20 mg and hyoscine 0.4 mg.

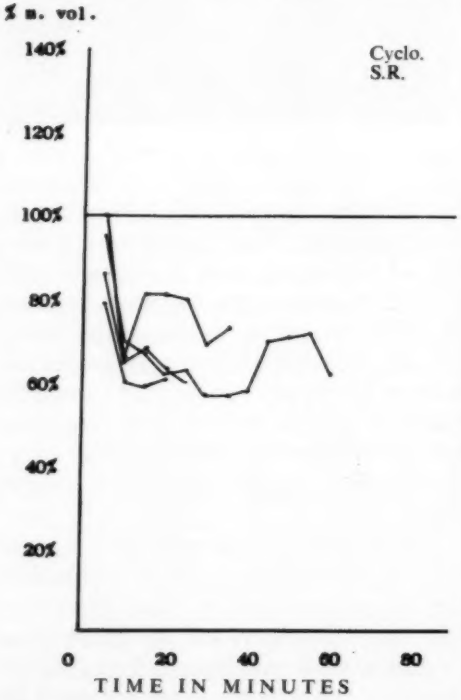

FIG. 3

Four patients: Thiopentone →Cyclopropane and Oxygen

Figure 4 shows that some depression was usual—initially it could be profound. From time to time a normal level of respiration was attained, but most remarkable was the great variability in the level of respiration. These variations may be due to surgical stimulation, but the relation has rarely been obvious.

Measurements were made during controlled respiration by several anaesthetists. Figure 5 shows the results obtained in eight patients. In this series all patients received a muscle relaxant, a light level of anaesthesia being maintained with nitrous oxide and oxygen following a thiopentone induction. It will be noted that ventilation was adequate in all cases (except at the conclusion), but that considerable variation occurred, hyperventilation attaining almost double the standard requirements in several instances.

This study thus revealed very wide variations in ventilation volumes in anaesthetized patients: with spontaneous respiration hypoventilation was usual; when artificial respiration was instituted the physiological requirement was often greatly exceeded. It is obvious that such variations in ventilation must lead to marked alterations in carbon dioxide levels. A further investigation was therefore undertaken to determine the magnitude of these changes and to correlate them with the observed ventilation.

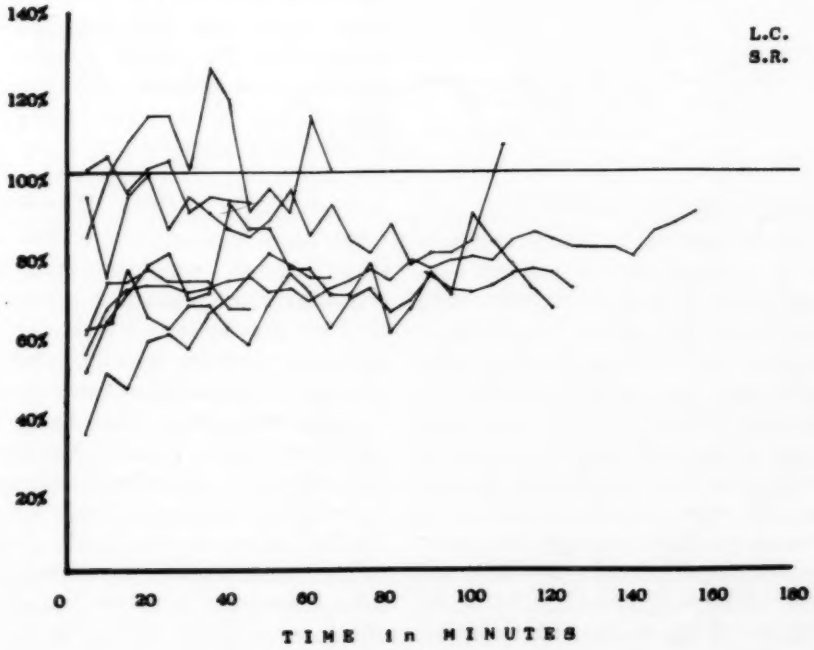

FIG. 4
Eight patients: "Lytic cocktail".

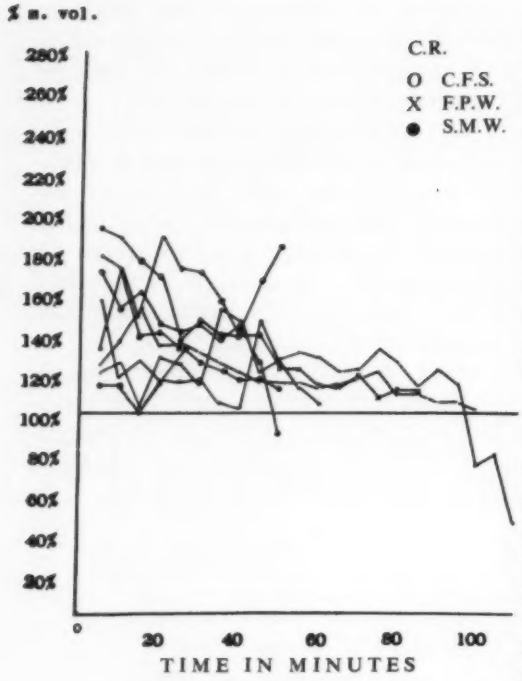

FIG. 5
Eight patients: controlled respiration.

CARBON DIOXIDE ESTIMATIONS

Various methods are available to obtain samples of alveolar gas for analysis: not all are suitable for use in anaesthetized patients. It was decided to use a simple method first described by Plesch (1909); the technique has been assessed by Boothby and Peabody (1914), who compared the results with those obtained by the Haldane method. Recently a method has been developed for direct analysis by the Draeger analyser of end-expiratory samples obtained from carinal level (Manley, 1956).

In the Plesch method a 500 ml rubber bag is filled with the anaesthetic gas mixture in use. By means of a short rubber connection controlled by a haemostat the bag is connected directly to the endotracheal tube in the patient. By manual pressure the bag contents are then driven to and from the lungs six to eight times in a period of less than fifteen seconds. This period, being less than one circulation-time, is insufficient to permit any marked change in gas concentrations in the blood flowing through the lungs. In this manner the contents of the bag are intimately mixed with alveolar gas, but it must be noted that the gas sample attains a state of equilibrium closer to mixed venous blood than to arterial blood (Peters and Van Slyke, 1932). In keeping with this, Boothby and Peabody (1914) pointed out that alveolar carbon dioxide tensions estimated in this way were about 4 mm higher than Haldane samples; this implies that the normal alveolar carbon dioxide concentration by the Plesch method will be just below 6 per cent. Immediately after collection the alveolar gas was analysed by means of a Draeger carbon dioxide analyser. Three analyses were made of each sample and a blank determination was performed on the anaesthetic gas mixture. It is important to perform the analyses without delay, because gas samples kept in a rubber bag change in composition, due to diffusion through the rubber. This change may be rapid when latex is used, but appears to be very much slower with the antistatic bag employed in these studies. The carbon dioxide concentrations have been recorded in graphical form against average ventilation (expressed as a percentage of the standard) observed during the ten-minute period immediately preceding sampling. The correlation between ventilation and

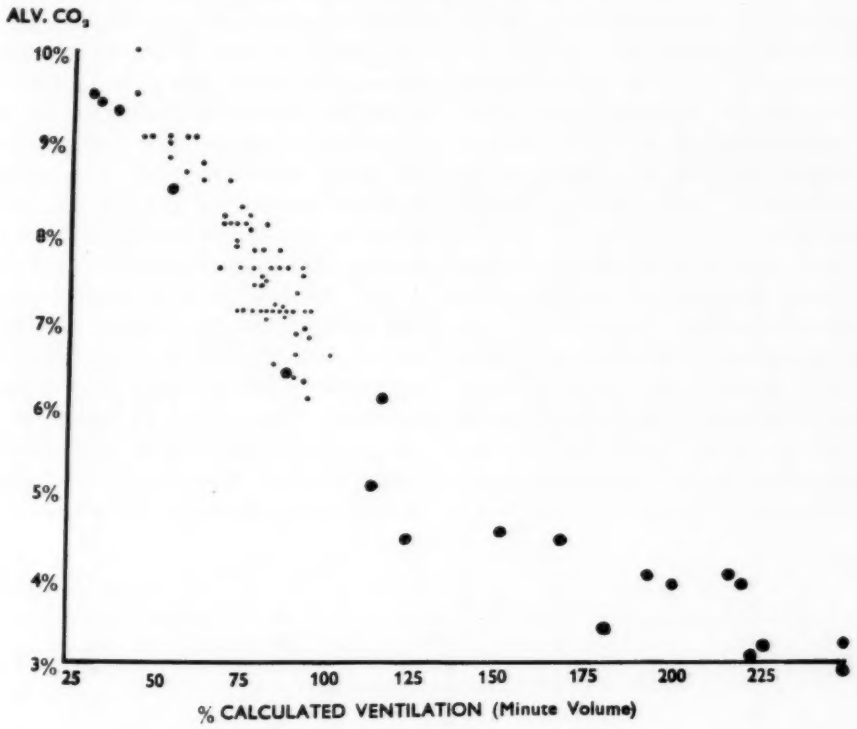

FIG. 6

Ordinate: Alveolar CO_2% (Plesch)
Abscissa: Average ventilation per minute (preceding 10 minutes) as percentage of standard.
• = Spontaneous respiration. o = Controlled respiration.

alveolar carbon dioxide concentrations is clearly seen. It is evident that very wide variations in alveolar carbon concentrations occur in anaesthetized patients. Note that all observations made during spontaneous respiration fall into the section denoting hypoventilation with elevation of carbon dioxide. Examination of the graph also shows that implementation of the standard ventilation will result in alveolar carbon dioxide levels close to the normal value.

It was previously pointed out that on theoretical grounds (Scurr, 1956; Comroe, 1955) absence of cyanosis could not act as a guide to carbon dioxide homeostasis, even in patients breathing air or 20 per cent oxygen with 80 per cent nitrous oxide. Observations were therefore made on a patient anaesthetized with 20 per cent oxygen and 80 per cent nitrous oxide to check ventilation volume, oxyhaemoglobin saturation (Stanco oximeter) and alveolar carbon dioxide level (Plesch). The results are shown graphically in figure 7. It will be noted that at twenty-one minutes the oxyhaemoglobin saturation fell below 80 per cent; only at this time was cyanosis

noted by visual observation. At other times during the anaesthetic raised alveolar carbon dioxide levels up to 9.5 per cent were noted, despite oxyhaemoglobin saturation well above the clinical cyanosis level. A series of these investigations has been performed with similar results (Jenkins, 1956).

At a later date observations were made on seventeen anaesthetized patients with the infrared analyser. The method used was similar to that described by Collier et al. (1955). These studies will be detailed in a later publication. The results obtained are shown in figure 8 and confirm that implementation of the standard ventilation assures near-normal alveolar carbon dioxide levels. During these studies it has been noted that it takes ten to twenty minutes for carbon dioxide equilibrium to be reached at the new level when the minute volume of ventilation is changed. Farhi and Rahn (1955) suggest, from work on animals, that 50 per cent of the change in carbon dioxide level is attained in four minutes, the readjustment proceeding in exponential fashion. With steady ventilation level, minor variations in alveolar carbon dioxide level are observed. These are probably due to uneven distribution in the lungs leading to variation in the sample (especially single end-expiratory samples). With regard to the total change in body stores of carbon dioxide, either by

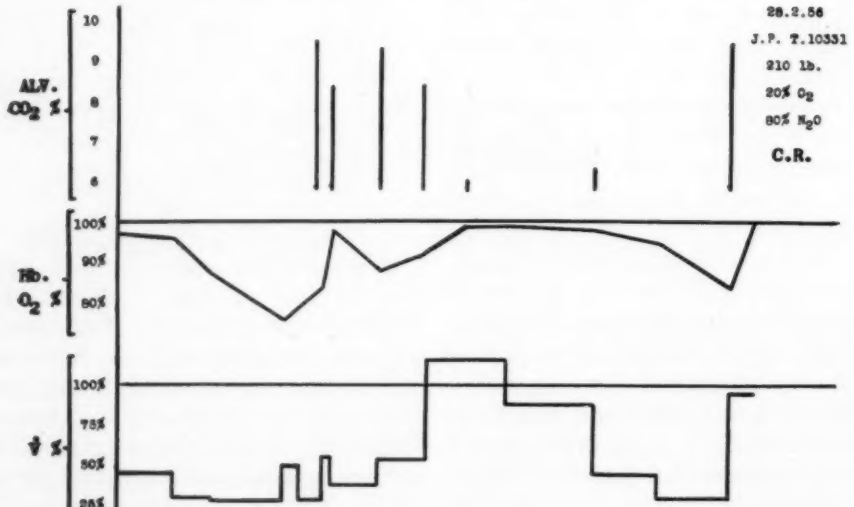

FIG. 7
Maintenance with 20% O_2:80% N_2O.
Alveolar CO_2% (Plesch).
Oxyhaemoglobin saturation (oximeter).
Ventilation % standard.

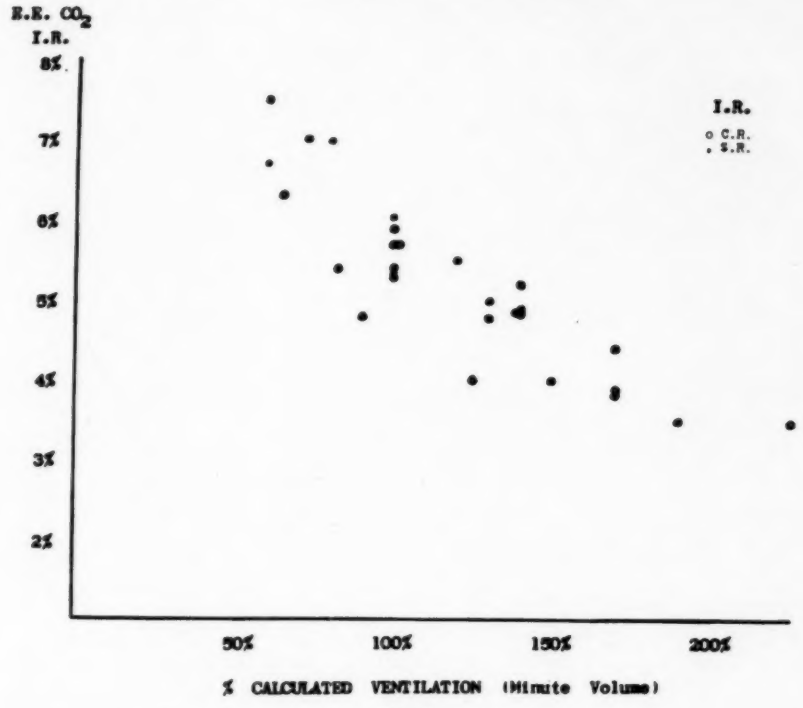

FIG. 8

Ordinate: Alveolar CO_2 % (Infrared analyser).
Abscissa: Average ventilation (preceding 10 minutes) as % of standard.

• = Spontaneous respiration.
o = Controlled respiration.

depletion or accumulation, Fahri and Rahn (1955) state that in dogs this is 1.5 ml per 1 kg body weight per 1 mm change in carbon dioxide tension. Work reported by Spurrell (1955) in man implies double this change in carbon dioxide volume.

It is concluded from these investigations that, provided carbon dioxide accumulation in the apparatus is prevented, it is possible to maintain alveolar carbon dioxide levels within normal limits by assuring a standard level of pulmonary ventilation, calculated from the patient's body weight. Under the usual conditions and in the dorsal position the arterial carbon dioxide tension is also likely to be normal, because close agreement between arterial and alveolar carbon dioxide tensions has been reported (Collier et al., 1955; Rahn and Suskind, 1954; Lambertsen, 1950; Buckley et al., 1952). When conditions are such that uneven gas distribution occurs in the lungs and when the perfusion-ventilation relations are disturbed (e.g. in the lateral position, pulmonary pathology or an open chest), ventilation requirements are likely to be

greater owing to factors leading to inequality of the alveolar and arterial carbon dioxide tensions.

SUMMARY

(1) Ventilation measurements have been made on anaesthetized patients: wide variations were observed, but spontaneous respiration almost invariably implied hypoventilation.

(2) Alveolar carbon dioxide concentrations were estimated and correlated with ventilation measurements.

Ventilation at the physiologically calculated standard rate assured near normality of the alveolar carbon dioxide concentration.

ACKNOWLEDGMENT

I am indebted to the British Oxygen Company's Research and Development Department for the loan and operation of the infrared gas analyser.

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FILM REVIEW

Artificial Respiration. I.C.I. Ltd., Film Library Cat. No. FA 4. Running time 11 mins. Black and white, 16 mm, sound.

This film is primarily intended for first-aid workers. The necessity of immediate artificial respiration in the treatment of all types of asphyxia is stressed and the Schafer and Holger Nielsen methods described and demonstrated by a team of first-aiders on a patient suffering from drowning. It is assumed that the operators know the mechanics of respiration, as the film is limited almost entirely to the technical performance of artificial respiration. The care in handling of the asphyxiated patient is excellent and emphasizes the importance of good team work when dealing with such an emergency. Furthermore it demonstrates how one can deal with such a situation singlehanded. Rather surprisingly, no comparison is made of the Schafer and Holger methods, but their combined use is demonstrated in detail.

Although frequent mention is made of the necessity of a clear airway, cleaning of debris from the mouth or pulling forward the tongue is not shown. These would probably be done automatically by a trained first-aider, but more emphasis could be placed on this aspect of treatment, for the benefit of the beginner. Mention might also be made of the use of inclined planes, such as might be found on a beach, to drain water from the lungs and bronchi.

The use of the "Novox" oxygen apparatus is shown in conjunction with artificial respiration. One only gets a fleeting glance at the machine and it is assumed that the cylinders are full and that the operator knows how to turn on the supply of gas. Perhaps it would have been better to omit entirely the use of oxygen, rather than leave one with the feeling that it is an extremely simple matter, with no dangers. Mention should have been made, at least, of the dangers of accumulation of vomitus or fluid into the mask which is strapped tightly to the patient's face.

This film will be extremely useful to physicians who lecture on first-aid, and is probably most useful at the conclusion of a talk during which the principles and indications for artificial respira- have been discussed. *John W. Dundee*

Brit. J. Anaesth. (1956), **28**, 431

CYCLOPROPANE INDUCTION FOR ANAESTHESIA IN DENTAL SURGERY

A Report on 800 *Cases*

BY

WILLIAM AULD

The Western Infirmary, Glasgow

NITROUS oxide continues to be the basis of anaesthesia for dental surgery. Despite the hazards associated with its use, there is scarce a dental surgery in the land wherein anaesthesia is not obtained by the use of nitrous oxide. It is the purpose of this paper to describe a technique which, it is thought, removes some of those hazards without contributing new ones.

The hazards are essentially those associated with anoxia, and, since anoxia in some degree is an essential concomitant of the production of anaesthesia by nitrous oxide (Macintosh, 1943; Clement, 1939), to avoid it means the use of an agent more powerful than nitrous oxide. There are many such agents; almost any other anaesthetic substance will provide anaesthesia in the presence of full oxygenation. But the dentist's requirements must be met, and these being quick induction, quick recovery and adequate anaesthesia, the words of Sir Robert Macintosh (1952) come to mind: "It is virtually impossible to fulfil those requirements with any anaesthetic. There is one condition, however, which gives rise to sudden unconsciousness, leads rapidly to a state approaching death and yet can be reversed almost equally rapidly—I refer to acute anoxia." Clement (1939): said: "The essentials for success are a minimum of oxygen during induction, time and a disregard for cyanosis."

The difficulties of nitrous oxide anaesthesia and the production of anoxia start with the induction. During induction it is the object of the anaesthetist so to reduce the oxygen supply to the brain that consciousness is lost. While achieving this the oxygen reservoir in the lungs and blood is diminished, and so obstruction, should it occur, produces a state of extreme anoxia almost at once. Also during induction difficulty may arise if the classical stage of delirium manifests itself too strongly, as it may in the apprehensive, the alcoholic and the physically strong. Breath holding may also occur and result in spastic movements. Induction in children may necessitate a very nice degree of judgment to obtain that state of fine oxygen balance which holds the patient between consciousness and circulatory failure. During induction there is a moment when the anoxic drive on the carotid body may cause the patient to take a great gasp of air by the mouth, thus wrecking a well planned anaesthetic. There is also a period during induction when "banging in the ears" may be experienced and may cause the patient to

gasp air or try to snatch off the mask. Some patients, too, become aware of the heart's beating and may, from fright, attempt to get out of the chair or otherwise interrupt the induction. Anaesthesia having been obtained, it may be found that masseter spasm exists in such degree that it is not possible to open the mouth with a gag without doing damage, and in some cases increasing the oxygen percentage to overcome this spasm produces instability in anaesthesia.

Clement (1939) writes: "The induction is usually the most difficult part of any anaesthesia." The induction of anaesthesia for dentistry can be just as difficult for one tooth as for twenty, and the degree of anoxia required to obtain surgical anaesthesia even for a short case may be extreme. Cyanosis develops after seven to twelve breaths of nitrous oxide, but the number of breaths before the patient is fit for surgery is a much more variable figure, and cases have been noted when as many as thirty breaths were required before the dentist could proceed. Using the technique to be described, the dentist can usually begin extracting after the tenth breath and, if the case be a small one, it may be completed without cyanosis having developed. It is an axiom that a smooth induction tends to produce smooth anaesthesia. Such an induction can be obtained with nitrous oxide in some cases if relatively large quantities of the gas are allowed to pour over the face. This does not remove the unpleasant sensations associated with increasing anoxia, however, though it does tend to diminish them, and the induction is slow. Clement (1939) advocates fast induction with pure nitrous oxide as a routine measure and states that by so doing the occurrence of excitement is diminished. The use of cyclopropane makes induction yet faster.

Cyclopropane is a powerful, nonirritating agent which can produce anaesthesia in a few breaths. It has been used by several workers to provide anaesthesia for dental surgery (Pinson, 1944; Gillies, 1948; Bourne, 1952; Marston, 1945; Clement, 1939). Save for Bourne, however, who is using it currently by a single dose technique, it seems to have fallen into disuse. One reason for this may be that if used as the principal anaesthetic agent it carries a high sickness rate: Bourne (1952) mentions vomiting as occurring in 21 per cent and nausea in 12 per cent of his series of 1,000 cases. Pinson (1944) also speaks of sickness being a disadvantage. Another reason is that delayed recovery may occur and one of the dentist's requirements is quick recovery. Yet a third reason is salivation and, fourthly, it is costly.

The technique to be described uses cyclopropane for induction only; maintenance being by nitrous oxide-oxygen, with or without trichlorethylene.

It has been found that with this technique the desiderata of safe and comfortable anaesthesia can be provided, viz.:

(1) Induction is so rapid that delirium does not occur.

(2) Breathing tends to be nasal because anaesthesia is established.

(3) Oxygen 8 per cent can be used for maintenance in the first instance.

(4) The masseters are relaxed and the mouth opens easily.

(5) The dentist's requirements are fulfilled.

The disadvantages of cyclopropane anaesthesia have not been seen, viz.:

(1) No vomiting or nausea.

(2) No salivation.

(3) No delayed recovery.

APPARATUS

Nitrous oxide being the basic anaesthetic agent, some form of pressure machine with intermittent flow is desirable. To this is added a cyclopropane flowmeter (fig. 1). The corrugated rubber breathing tube is replaced by a tube of 300 ml capacity and is suitably obtained by a tube of 2 cm inner diameter and 95 cm length. This tube permits of free respiration. The large rubber bag of the Magill rebreathing attachment is replaced by one of 500 ml capacity having a stopcock at its lower end. The cyclopropane flowmeter and the stopcock are connected by a rubber tube. A watch or other timing device is required to measure one minute.

TECHNIQUE

The small rubber bag having been charged with 500 ml of cyclopropane it is

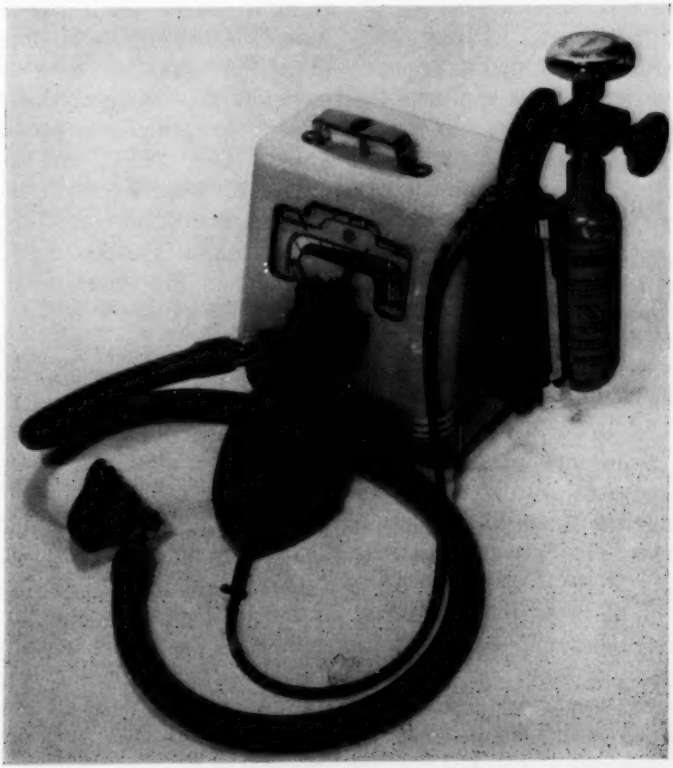

FIG. 1
The apparatus.

then closed. For children under the age of ten years, 350 ml of the gas are used. Induction is started with nitrous oxide at low pressure. In the average adult five breaths of nitrous oxide are permitted, the expiratory valve being open. On the sixth inspiration the bag is opened and remains so for two breaths. If the first breath does not empty the bag owing to small tidal air, it should be emptied manually during the second breath and then closed. Within a few seconds the patient becomes unconscious as shown by the onset of automatic breathing and relaxation of the muscles, and the oxygen percentage can be immediately turned up to 8 per cent. If it is to be a long case, trichlorethylene can be added if desired. It will be found that the jaw is pleasantly relaxed. Thereafter anaesthesia is maintained according to one's custom. Recovery is rapid. Some of the unpleasantness of recovery from an anoxic interlude is avoided; it is said in one clinic, where young children are seen in large numbers and cared for by professional nurses, that there is less crying in the recovery room when this technique has been used. Patients who have experienced both forms of induction have expressed a preference for that using cyclopropane. Sickness has occurred in two cases in this series, both patients having had food shortly before presenting themselves. Salivation has not been noted.

The types of cases in this series are representative of most practices. The ages vary from three years to eighty years, the number of teeth to be extracted at one visit, from one to thirty, and the type of patient from those met with in a West End practice to those seen in Dockland.

DISCUSSION

The successful employment of this technique depends upon the capacity of the bag and breathing tube. If the tube or bag is too big, insufficient cyclopropane reaches the patient in one breath and too great a quantity of the gas is expelled via the expiratory valve on the subsequent expiration. The capacities of the bag and tube, 500 ml and 300 ml respectively, are not arbitrary figures, but bear a relationship to the tidal air. The average adult tends to increase his tidal air during nitrous oxide induction and 800 ml to 1,000 ml is a not unusual amount. That being so, on the sixth inspiration there is inhaled 1,000 ml of a 50 per cent mixture of cyclopropane and nitrous oxide. This is below the concentration necessary to make it irritating or to produce laryngeal spasm (Lee, 1953), and it follows the advice of Waters (1945), who advocates the rapid addition of cyclopropane to the inhaled gases. The concentration of cyclopropane in the lungs if full inspiration occurs will be about 8 per cent after the sixth breath. Absorption and dilution by subsequent breaths will reduce the concentration until, by the twelfth exhalation the amount of cyclopropane in the exhaled gas is about 1.5 per cent which is below the explosive limit (Lee, 1953), and it is at this point that the dentist usually commences his work.

This technique has been used for patients of all ages, and except for those under ten, no modification in the amount of cyclopropane is made. It is thought that by ensuring that the expiratory valve is open, the quantity of cyclopropane inhaled is limited by the tidal air: if the tidal air is large, 500 ml. of cyclopropane are

received. If it is small, a correspondingly smaller quantity is inhaled and each expiration ejects some fresh gas from the tube and reduces the total amount received. The continued flow of nitrous oxide under low pressure also assists in this.

It is interesting to observe that despite the expense of cyclopropane, the cost per case is a penny or two less than when nitrous oxide alone is used. This is explained by the diminished consumption of nitrous oxide, which is reduced by half. When used alone, 100 gallons of nitrous oxide sufficed for seven cases; used with cyclopropane induction, fourteen cases is the average figure. The great saving in nitrous oxide occurs during induction.

ACKNOWLEDGMENTS

I am indebted to Mr. Kirkwood of the British Oxygen Company, Glasgow, for designing and constructing the cyclopropane bracket for attachment to the Devanaest; to Mr. James L. A. Evatt, A.I.B.P., A.R.P.S., F.S.A.Scot., of the Department of Medical Photography, Royal Hospital for Sick Children, Glasgow, for the photograph; and to the anonymous dentists who tolerated and then encouraged the initial experimental work.

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BOOK REVIEW

Anesthesia—Thief of Pain. By Sylvan M. Shane, D.D.S. Published by Vantage Press, Inc., 120 West 31st Street, New York 1, 1956. Illustrated. Price £1.

Dr. Sylvan M. Shane has written *Anesthesia—Thief of Pain* to dispel the fear and apprehension often associated with anaesthesia. The morbidly curious and some stoics may like this book, but individuals vary and the effect may well be the reverse from what is intended.

The patient who is to have a spinal anaesthetic and learns that he will see surgical instruments for use on himself is going to flinch, but even the staunchest is likely to quail if he thinks he may get a mirror view of the operation in progress. Surely this is not the way to conduct spinal anaesthesia.

The graphic account of a woman crying out piteously in labour is hardly calculated to inspire confidence, and readers will have some misgivings when they find the anaesthetist appeared indifferent, dismissing the situation thus: "Actually, the patient is *often* not experiencing the pain she complains of, since she has been given sedatives and is quite amnesic." The italics are mine. The meaning of the word amnesic can be inferred later. There is no index.

The description of sensations perceived during the induction of inhalational anaesthesia culminating with a "bang" may cause some patients to eschew this method.

Clearly his Doctor Payneless is unfamiliar with routine relaxant practice, or is it that the author thinks this information should not be divulged?

There is no doubt that patients should be informed about what they are to expect, but the knowledge gained from reading this book will make some want to be in at the kill; this may be embarrassing.

Though parts of the book are excellent and it makes amusing reading, I should not recommend it to patients as a whole; they have to face something which cannot be laughed aside, and if they really want to know, the truth can be explained to them individually, more palatably, and without causing anxiety to others. Herein lies a part of the skill our art requires.

R. P. Harbord

FACULTY OF ANAESTHETISTS

OF THE

ROYAL COLLEGE OF SURGEONS OF ENGLAND

The following candidates were successful in the examination for Diploma in Anaesthetics (May 1956):

Ainley-Walker, J. C. S.
Anderson, J. L.
Brandstater, B. J.
Burkinshaw, Daphne
D'Bras, B. E. G.
Delaney, E. J.
French, O. H.
Genever, R. J.
Jolly, C. R.
Kaufman, L.
Keen, R. I.
Laurie Smith, N.
Lumb, Patricia Mary.
Marshall, M.
Merrifield, A. J.
Parker, Sheila Margaret.
Phillips, A. H.
Poon, Yee Kit
Prince-White, Freda Elizabeth.
Rolfe, M. G.
Schooling, Isabel Brenda.
Sims, A. J.
Thomson, T.
Webster, A. C.
Wise, R. P.
Wright, D. G. R.
Young, T. M.

The following candidates were successful in the Primary examination of Fellowship in the Faculty of Anaesthetists (June 1956):

Bastable, Morag Joyce Rankine
Booth, A. J.
Boutros, Azmy Ragheb
Braude, W.
Campkin, T. V.
Carroll, G. J.
Chari, J.
Coutanceau, H. R. P.
Cross, J. R.
Davidson, J. T.
Davies, T. D. W.
Dinmore, P.
Giles, R. C.
Hurter, D. G.
Irvine, Mary Clarenza
Keddie, F. S.
Kemp, S. W.
King, R.
Langdon-Herring, L.
Lau, G. L. F.
Lester, E. R.
McClelland, R. M. A.
Mathie, Angela
Medeau, Marie Madeleine
Mehta, Prabhakar Mayabhai
Montgomery, J. B.
Mortell, Annie
Ozinsky, J.
Paton, A. S.
Plantevin, Odile Maire Emma Paule
Pryor, C. H.
Purkis, I. E.
Sloan, I. A.
Stark, D. C. C.
Verrill, P. J.
Walmsley, D. A.
Ward, C. S.
Wilkinson, W. M.

SCIENTIFIC MEETING

211 persons attended the Scientific Meeting held at the Faculty of Anaesthetists of the Royal College of Surgeons on Saturday, May 5, 1956, the programme of which was as follows:

"The Physiology of the Adrenal Gland" by Professor J. H. Burn, D.M., F.R.S.

"Adrenocortical Syndromes" by Dr. A. W. Spence, F.R.C.P.

"The Use of Cortisone in Surgery" by Dr. E. F. Scowen, F.R.C.P.

"Anaesthesia and the Adrenals" by Professor W. W. Mushin, F.F.A.R.C.S.

FUTURE COURSES OF LECTURES ON ANAESTHETICS

It has been decided to hold three two-week full time Lecture courses a year instead of two three-week ones as was the case previously. The Autumn course will be held from October 15 to 26 and will consist of 32 lectures; nightly discussion groups and visits to London hospitals for medical clinical conferences and demonstrations in practical anaesthesia.

Fees: Lectures £12 12*s*.
Discussion Groups £10 10*s*.
Clinical Conferences £2 2*s*.

A two-week course of evening lectures in Pharmacology will be held from November 26 to December 7. Fee £6 6*s*.

FILM REVIEW

Examination of an Unconscious Patient. I.C.I. Ltd., Film Library Cat. No. FA5. Running time 6 mins. Black and White, 16 mm, sound.

The catalogue gives the object of this film as being "how to teach first-aiders to use a systematic method of examination". In this respect it is excellent, even though the commentator uses such terms as "sterterous" and "airway", neither of which is likely to be understood by non-trained first-aiders. The rapidity with which the various parts of the body are examined is such that a nonmedical audience would need to see the film several times before they could assimilate all it contains. Since it only lasts six minutes this is not a serious objection.

It seems rather peculiar to see the attendant cover up an unconscious patient and leave him lying on his back, open to all the dangers of aspiration of vomitus. Since no broken bones were found in the patient, this would have been an excellent opportunity of demonstrating the use of the "tonsil position", the safety of which is not always appreciated, even by physicians.

John W. Dundee

SCOTTISH SOCIETY OF ANAESTHETISTS

The Scottish Society of Anaesthetists held its Annual Meeting at Dunblane Hotel Hydro, Perthshire, from Friday, April 27, 1956, until Sunday, April 29, 1956. A company of seventy members, their wives and guests, was present.

The Executive Council for 1956–57 is as follows:

President Dr. H. Bruce Wilson, Aberdeen.

Vice-President Dr. R. Lawrie, Perth.

Hon. Secretary Dr. A. G. Miller, Glasgow.

Dr. F. G. Gibbs, Edinburgh.
Dr. J. W. Levack, Aberdeen.
Dr. F. Holmes, Edinburgh.
Dr. W. Auld, Glasgow.
Dr. J. R. Kyles, Edinburgh.
Dr. W. Christie, Dundee.
Dr. H. Fairlie, Glasgow.

Dr. H. Bruce Wilson in his Presidential address, entitled " Widening Horizons ", outlined some of the problems in which the anaesthetist might be invited to co-operate outwith the actual technical administration of anaesthetics. These were listed under seven headings:

(1) The treatment of bronchial tree obstruction, both postoperative and in cases with neurological lesions involving the bulb.
(2) The treatment of cerebral vascular catastrophies.
(3) The assessment of peripheral vascular lesions.
(4) The treatment of cases of paralytic poliomyelitis and severe tetanus.
(5) Treatment of overdosage with hypnotic, sedative and analgesic drugs.
(6) Association with the physicist in evolving equipment for use in anaesthesia and research.
(7) Hypnosis.

Dr. Bruce Wilson then went on to consider each of these headings in detail, stressing especially the value of co-operation by the anaesthetist in treatment of cases of paralytic poliomyelitis, bronchial tree obstruction, and various forms of poisoning.

Dr. M. D. Nosworthy, London, was the guest of the Society and gave a most interesting and practical talk on the dangers associated with reflex stimuli, both anaesthetic and surgical. He advised that an adequate period should always be allowed for a muscle relaxant to take full effect before intubating any patient and drew attention to the possibility that postoperative atelectasis was a sequel in many cases to bronchial spasm. He preferred cyclopropane to nitrous oxide for maintenance of anaesthesia, as the light level of of narcosis with the latter could allow surgical stimuli to produce cardiovascular depression. In cases where a nonexplosive agent was necessary, Dr. Nosworthy used adequate doses of pethidine after induction of anaesthesia and before the operation was started.

The prize which the Scottish Society of Anaesthetists awards annually for the best paper submitted by an anaesthetist of the Senior Registrar grading or below was won by Dr. David B. Murray, Glasgow, with a paper in which he compared the actions and uses of Buthalitone Sodium and Thiopentone Sodium.

CORRECTION

Messrs. Roche Products Limited have been kind enough to point out that in a report of a meeting of the Society of Anaesthetists of the South-Western Region, published on page 239 of the May issue, the approved name of Arfonad was given as " Verazide." This is, in fact, incorrect, the approved name for Arfonad being trimetaphan (camphorsulphonate).

We apologize for this error and hope that no inconvenience has been caused.

BOOK REVIEW

Psychosomatic Aspects of Surgery. Edited by Alfred J. Cantor, M.D., and Arthur N. Fox, M.D. Published by Grune & Stratton, New York, 1956.

This is an account of the first annual meeting of the Academy of Psychosomatic Medicine, held in New York in October 1954. *Mens sana in corpore sano* is an ideal to which we all strive; it is also a statement that can reasonably be made about any healthy man or woman. But when the body is injured by accident or disease, bearing in mind that when one member suffers all the members suffer with it, there must be a psychic counterpart, and vice versa. This surely is a commonplace of medicine, or as Dr. Cantor puts it "Psychosomatic medicine is medicine."

Dr. Rovenstine contributes the article on anaesthesia and naturally in this symposium stresses the desirability of the anaesthetist making himself the "Guide, philosopher and friend" of the patient to the utmost of his capacity or for all he is worth. In no field of anaesthesia does this apply so much as in that of spinal anaesthesia. He quotes from some unnamed author as follows: "The aim of premedication is to produce an untroubled, serene, drowsy and relaxed patient; to reduce alarm responses and reflexes to dry secretions and to protect against vagal insults. . . . In addition the agent should have no side effects and be otherwise nontoxic. Chlorpromazine appears to be such an agent". But surely these are the effects that morphia and atropine have produced in millions of cases in the last half-century. Yet Dr. Rovenstine goes on to say, "The age-old, time-honoured pre-anaesthetic medicants such as morphine, atropine, etc., are to be practically abandoned."

Dr. B. B. Raginsky who contributes a second article on psychosomatic aspects of anaesthesia again stresses the importance of the personality of the anaesthetist but apparently disagrees with Dr. Rovenstine on the importance of chlorpromazine, for he says, "Nothing remarkable has been discovered in the last few decades in the field of chemotherapy for this condition" (pre-anaesthetic condition). Dr. Raginsky says. "The most forceful method of allaying anxiety without resort to medication is in the use of hypnosis." But it must be in capable hands.

No one would deny that surgeons do a great amount of good in the world. It does them no harm perhaps to be told by the psychiatrist that though their operations can remove an appendix they do not always get rid of the emotional disturbance that has gone with it; nor do they cure what is primarily psychologic disturbance by operation, however many. That people who look at the human being from such opposite poles as the psychiatrist and the surgeon should meet together and discuss the subject, the human being, is probably a good thing and gives to both a a broader view of the matter and of each other. In a collection of papers such as this, not only are various points of view apparent but the language in which they are expressed is still more varied. For example: "The diagnosis of a cardiovascular lesion needing surgical intervention must be made accurately, and must be as devoid of error as is humanly possible." Here is a plain statement in English, but what is this? "Such a hololologic approach, i.e. from the viewpoint of the logic of the whole person, leads us to deal with a person's 'total' biosocial integration and not only with his presenting somatic symptom(s). The presenting somatic problem does not necessarily represent his only, and often not even his main problem. But even if it would, its analysis and evaluation against the background of his integrational stress constellation will yield therapeutically more exploitable information. The inter-relatedness of the aforementioned six psychodynamically significant biosocial integrational factors can be expressed in the following quasi-mathematic formula:

$$SQ = \frac{(G+T)O}{(E+M)S} = \frac{(\text{Goals} + \text{Tasks})\ \text{Obstacles}}{(\text{Equipment} + \text{Means})\ \text{Striving}}$$

There follows a definition of the symbols.

E. Falkner Hill

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